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GUY'S HOSPITAL  
REPORTS.

EDITED BY  
SAMUEL WILKS, M.D. LONDIN.,  
AND  
ALFRED POLAND.

Third Series.

VOL. II.



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September, 1857.



# MYELOID TUMOUR OF THE SCAPULA.

---

BY EDWARD COCK.

WITH A DESCRIPTION OF THE GROWTH,

BY SAMUEL WILKS, M.D.

---

E. C—, æt. 27, admitted, under Mr. Cock's care, into Dorcas Ward, on January 16th, 1856, for a tumour on the left shoulder. She was a remarkably small, delicately made woman, but her health had always been good; and on admission she was suckling her third child, then five weeks old. About a year and nine months before this time, she began to feel uneasiness and occasional pain in the region of the left shoulder, which prevented her lying upon it, but she was not aware that any swelling of the part existed, until in July, 1855 (six months preceding admission), when she fell and struck it. She then perceived a lump growing, according to her description, from the spine of the scapula, and as this seemed to increase in size, she applied, in the month of August, at the Middlesex Hospital for advice, where lotions and iodine were applied; removal of the tumour was also recommended, but postponed on account of her pregnancy, which was then advanced about five months. After this, the tumour rapidly grew, and particularly lately. She had a good confinement, and a month afterwards came into the hospital. A large tumour, the size of a foetal head, was seen to project from the left shoulder. It evidently sprung from the spine of the scapula, and it took the form of the shoulder, whose position it completely occupied. It was covered by the deltoid muscle, the fibres of which were expanded over it, and nearly defined its situation and extent. The skin covering it was pale; it

had thus a tolerably uniform and rounded appearance, with the exception of a projecting nodule which sprung from its front and inner side. When handled it felt very hard, and was evidently composed externally of bony tissue, but from the impossibility of knowing its internal structure, Mr. Cock was content to give it the general name, osteosarcoma, considering that, in all probability, it was enchondromatous, to which, indeed, it was allied.

On January 22d, the patient was placed under the influence of chloroform, and Mr. Cock removed the tumour. Upon dissecting off the skin, and cutting through the deltoid muscle, it was found to be growing from the spinous process of the scapula, so that in order to entirely detach it, it was necessary to remove the greater part of this process, from which it had been developed, and likewise to snip off the acromion end of the clavicle. The wound healed favorably, and the patient left the hospital well, on March 1st, and up to the present time has remained in good health.

The tumour when removed, was found to weigh nearly a pound, and to measure in its long circumference, 12 inches, and in its short, 10 inches. It was of an oval shape, having, however, that side concave, which constituted its base, and which covered the shoulder. This inferior surface, and the sides immediately springing from it, were bony, so that the greater part of the tumour was enclosed in an osseous shell. The acromion end of the clavicle which had been removed, still articulated with an eminence of bone corresponding to the acromion itself, and the latter was gradually lost in the osseous laminæ which formed the wall of the tumour. It was thus clearly seen, that the growth was derived from an expansion of the spine of the scapula itself, and which, indeed, formed its sides; the most superficial part only of the tumour being soft, and its walls consisting of fibrous membrane.

Upon making a section of the tumour, a brain-like mass was exposed, inclosed in a thin-walled cyst, the walls being about one eighth of an inch thick, and bony, with the exception of the most superficial part, which was membranous. Upon closer examination, the internal substance was seen to be composed of an opaque white, curdy structure, and a pink, gelatinous-looking material. The former largely predominated.

This was in consistence and colour something like cream-cheese, or blanchmange, and therefore, firmer, drier, and more friable than medullary cancer, and was not capable of emitting any juice, or being formed into a paste by squeezing, as in the latter.

It was, however, not unlike the appearance presented sometimes by *dead* cancer. The other and characteristic element of the tumour existed only in a minor proportion, and was scattered throughout that just mentioned, in the form of small masses each about the size of a pea. These were of gelatinous consistence of a slight translucent appearance, and of a dull pinkish colour, this colour being altogether peculiar, and one not very often met with either in healthy or morbid structures. The hue was in no way due to blood, for no corpuscles or other elements of this fluid were discoverable. After being in spirit a short time, the colour for the most part, disappeared. The whole of the contents of the tumour were very friable, and portions fell out upon inverting it. (See Plate I.)

*Microscopic examination.*—The white matter was composed principally of fatty granule masses, amongst which were seen the many nucleated cells which composed the entire mass of the translucent portions; from which we conclude that this fat was a superaddition to, or the result of a degeneration of, the other previously existing elements. If this be true, the original structure consisted wholly of the pink semi-transparent parts which were now found to occupy only a minor portion of the tumour; and, therefore, if the latter had been seen at an earlier period, its whole appearance and composition would have had this character. At the time of removal, however, this was being fast destroyed by a fatty degeneration. The elements of which we speak, composing this part, consisted of the mother-cells, described by Lebert as characteristic of one of his forms of fibro-plastic growths, which peculiarity has led Paget, with good reason, to separate those tumours which possess them into another class, which he calls “myeloid,” a designation founded upon the resemblance of the cells to those existing in the *marrow* of the foetal bones.

The cells in this tumour corresponded in most respects with those described by the above-named authors, excepting



that the cells were much larger, and contained many more nuclei, some cells holding at least a hundred. Many of the cells, too, were sprouting, sending out processes in all directions, from their sides, which joined and interlocked with each other. It will be seen by the drawing that these cells were of all shapes and sizes, some being small, and others containing only one or two nuclei. (See Plate V, fig. 4.)

The interest of this case lies not so much in its rarity, for no doubt many similar ones may be found in the hospital museums, as in the fact that this description of tumour has only of late been fully recognised and described; all growths from bone having been indiscriminately classed under the general name of osteo-sarcoma. Lebert, many years ago, described, amongst his fibro-plastic growths, some which contained the peculiar cells observed in the present instance, but only recently has Paget shown that some are wholly composed of these elements, and are therefore deserving of another name, "myeloid." There is, undoubtedly, a natural distinction between a hard fibrous tumour and a soft one consisting of cells, and therefore a division was requisite; at the same time, the confusion which Lebert was supposed to make with respect to their constitution, is one which really exists in nature, for the two forms are very constantly found in combination. In such a mixed tumour, the presence of myeloid cells is not sufficient to determine its designation as myeloid, for, by so doing, we gain a wrong impression of the growth, both in its anatomical and pathological characters. For example, this compound structure is seen in the tumour from the gum, below mentioned, which is harder than the true myeloid, and is disposed to return. And this suggests an important question with respect to the growth under discussion; is it malignant, and has it a disposition to reappear after removal? All experience gained at present with respect to these growths, would favour the answer that they do not return,—at least, this is true of the tumour in its pure myeloid form,—the present instance affording a very good example of their history in general, the main points of which are, that they occur in young persons, are isolated, grow from bone, and do not return on removal. The whole question of malignancy, however, is still in abeyance, and appears at present to be only a comparative



term, since we continually see simple fibre tissue reproduced after removal, and then acquire the epithet malignant; in the same way osseous growths, from the tendency to develop themselves in a variety of parts, have caused us to recognise a class of diseases known as osteoid cancer, and more lately we have witnessed simple cartilage developing itself in the lymphatic gland, lungs, &c., to the utter destruction of the patient, in the same way as ordinary cancer would have done. The simplest structures, therefore, are not free from the taint of malignancy, using the term in its most general signification. Looking, however, upon myeloid tumours with reference to the question in its older and more restricted sense, we should certainly say that their elements are as benignant as nucleated fibre, bone, cartilage, &c., to which, indeed, they are closely allied. In all probability, however, the harmless and the more destructive elements may be combined; for, as bone may be developed together with cancer, as is seen in osteoid growths, and as cartilage may be developed with cancer, as has been seen in the testis, so, no doubt, myeloid matter may coexist with cancerous elements. In the figures given by Wedl, in his '*Pathological Histology*,' of cancer of bone, there are some many-nucleated cells, evidently of the myeloid class, although he does not adopt this name.

It may not be uninteresting to observe that, during the period in which this tumour was being so rapidly developed on the shoulder, similar structures to those which it contained were being as quickly produced in the fœtal structures in the uterus of the patient; she being at the time pregnant.

---

#### APPENDIX.

##### EPULIS FROM GUM OF A MYELO-FIBROUS CHARACTER.

Catherine F—, æt. 23, was admitted under Mr. Birkett's care, in February, 1856, for a tumour growing from the gum of the lower jaw, between the tricuspid teeth. The girl was a servant, in good health, and the growth had been coming fourteen months. Upon removal and examination, it was

found to be very tough, and was composed of fibro-plastic matter, amongst which were numerous myeloid cells. The latter exactly resembled those already described in Mr. Cock's case. (See Plate V, fig. 5.)

A growth from the gum or epulis was given to me (Dr. Wilks) some years ago, by Mr. Cooper Forster, which he had removed from a lady, and which exactly resembled the above in every respect, consisting of fibres and mother-cells holding many nuclei. It had been growing three years, and had been removed once before. Mr. Forster tells me it has again returned.

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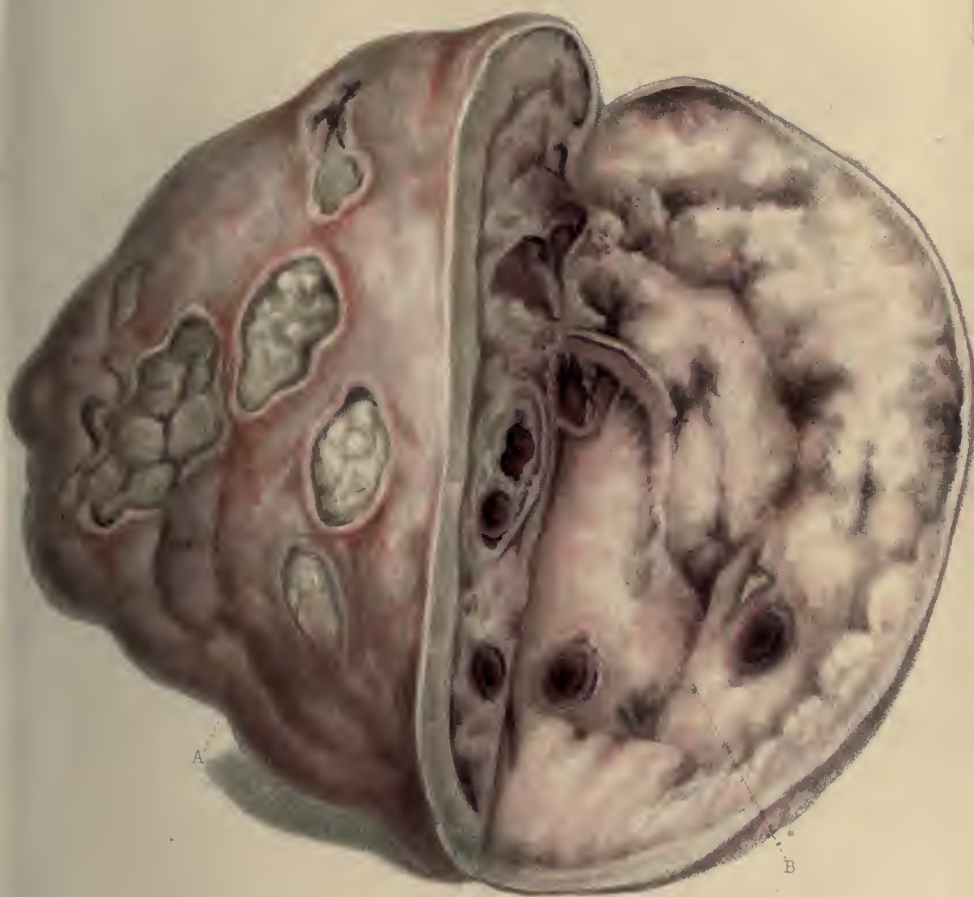
## PLATE I.

REPRESENTS THE MYELOID TUMOUR CUT OPEN.

A. The osseous shell.

B. The internal structure, exhibiting the peculiar myeloid matter.

Plate I.





# THIRD SEPTENNIAL REPORT

OF

GUY'S LYING-IN CHARITY,

FROM OCTOBER 1, 1847, TO OCTOBER 1, 1854.

ALSO

REPORT OF THE LYING-IN CHARITY FOR  
TWENTY-ONE YEARS,

FROM OCTOBER, 1, 1833, TO OCTOBER 1, 1854.

COLLATED FROM THE RECORDS,

BY SERGEANT J. C. NORMAN,

LATE RESIDENT OBSTETRIC CLERK.

PRESENTED

BY J. C. W. LEVER, M.D., AND H. OLDHAM, M.D.

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## INTRODUCTION.

IN presenting the following Reports of the Lying-in Charity of Guy's Hospital, the latter of which embraces a period of twenty-one years, to our readers, it is necessary to remark that the Institution, which was established by the late Mr. Harrison, is designed for the instruction of the pupils in practical midwifery, and that the patients are attended at their own houses. Three pupils—two of whom are senior men, and have obtained legal qualification to practise—are appointed resident obstetric clerks, living within the walls of the hospital at the expense of the charity, and ready at all times to assist with their counsel and cooperation the body of junior students who may be attending cases. These offices, which are purposely invested with a good deal of responsibility, are of great value to their possessors, from the opportunity which is afforded them to witness and manage difficult cases of midwifery, not even



excepting instrumental cases, and it affords us much pleasure to record our general satisfaction with the industry and good conduct which have animated the great body of students during this long period, in discharging the duties which have devolved upon them.

We are aware, however, that the Report bears indications of the kind of inexperience with which its details have necessarily been narrated, and our wish has been not to strain its deductions too far, or to record niceties of practice which can alone be safely derived from more experienced practitioners.

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## PART I.

### THIRD SEPTENNIAL REPORT OF THE LYING-IN CHARITY.

From 1847 to 1854, 11,224 women have been attended by the pupils attached to the Lying-in Charity.

The following Table (I) will show the number of women confined in each month during the seven years.

TABLE I.

*Showing the Number of Women confined in each month in Seven Years, extending from 1847 to 1854.*

Months.	1847. 1848.	1848. 1849.	1849. 1850.	1850. 1851.	1851. 1852.	1852. 1853.	1853. 1854.	Total.	Ratio per Cent.
October	97	103	123	137	138	157	154	909	8.09
November	86	148	110	130	153	163	134	924	8.23
December	100	134	129	142	152	151	138	946	8.42
January	90	130	113	154	161	189	146	983	8.76
February	116	142	118	157	144	157	178	1012	9.01
March	88	119	135	169	185	183	168	1047	9.32
April	90	108	111	154	142	147	132	884	7.87
May	104	115	112	127	138	153	132	881	7.87
June	96	91	138	132	162	130	128	877	7.87
July	111	118	134	134	135	159	144	935	8.24
August	92	105	117	154	120	161	147	896	8.09
September	119	104	110	135	149	162	151	930	8.23
Total . .	1189	1417	1450	1725	1779	1912	1752	11224	100.00

By comparing the totals in this table it will be seen that in *March* the greatest number of confinements have taken place, and in *June* the least. The months, in this respect, stand in the following order :

March.	December.	November.	April.
February.	July.	October.	May.
January.	September.	August.	June.

Table II shows the number of the confinement in 11,224 cases, extending from 1847 to 1854.

By examining this table, it will be seen that the greatest number of women have been attended in their second confinement, and that (with one or two exceptions) the number gradually lessens as the repetitions of pregnancy increase; thus it will be seen, that while 1845 women were attended in their second confinement, there were only 1723 in their first, one in the 23d, and none in the 21st, 22d, and 24th.

TABLE II.

*Distinguishing the Number of the Confinement in 11,224 Cases extending from October, 1847, to October, 1854.*

Number of Confinement.	1847. 1848.	1848. 1849.	1849. 1850.	1850. 1851.	1851. 1852.	1852. 1853.	1853. 1854.	Total.	Ratio per Cent.
First . . .	186	204	202	287	300	295	249	1723	15·35
Second . . .	205	240	235	261	300	300	304	1845	16·43
Third . . .	164	201	186	268	254	274	275	1622	14·45
Fourth . . .	153	188	208	190	207	260	236	1442	12·84
Fifth . . .	108	162	155	183	193	207	177	1185	10·55
Sixth . . .	82	116	125	168	132	164	159	946	8·42
Seventh . . .	109	103	103	128	101	131	129	804	7·08
Eighth . . .	68	65	93	87	93	97	65	568	5·06
Ninth . . .	43	52	66	55	70	75	62	423	3·75
Tenth . . .	34	42	35	46	46	54	43	300	2·67
Eleventh . . .	21	19	19	29	37	25	27	177	1·57
Twelfth . . .	7	5	6	9	25	15	14	81	0·72
Thirteenth . . .	2	9	3	4	10	3	4	35	0·31
Fourteenth . . .	4	2	5	4	3	8	6	32	0·28
Fifteenth . . .	2	5	5	1	5	3	—	21	0·18
Sixteenth . . .	—	1	—	1	1	—	1	4	0·03
Seventeenth . . .	—	1	1	1	2	1	—	6	0·05
Eighteenth . . .	—	1	—	1	—	—	1	3	0·02
Nineteenth . . .	—	—	1	1	—	—	—	2	0·01
Twentieth . . .	—	1	2	1	—	—	—	4	0·03
Twenty-first . . .	—	—	—	—	—	—	—	—	—
Twenty-second . . .	—	—	—	—	—	—	—	—	—
Twenty-third . . .	1	—	—	—	—	—	—	1	0·00
Twenty-fourth . . .	—	—	—	—	—	—	—	—	—
Total . . .	1189	1417	1450	1725	1779	1912	1752	11224	100·00

Table III distinguishes the sex in 10,816 children born *alive*; and Table IV distinguishes the sex in 512 children *still-born*.

TABLE III.

*Distinguishing the Sex of 10,816 born alive.*

Sex.	1847.	1848.	1849.	1850.	1851.	1852.	1853.	Total.	Ratio per Cent.
	1848.	1849.	1850.	1851.	1852.	1853.	1854.		
Males . .	589	714	722	829	936	978	902	5670	52·42
Females .	553	653	667	834	772	867	800	5146	47·58
Total .	1142	1367	1389	1663	1708	1845	1702	10816	100·00

TABLE IV.

*Distinguishing the Sex in 512 still-born children.*

Sex.	1847.	1848.	1849.	1850.	1851.	1852.	1853.	Total.	Ratio per Cent.
	1848.	1849.	1850.	1851.	1852.	1853.	1854.		
Males . .	42	46	47	51	54	56	38	334	65·23
Females .	17	22	25	23	39	28	24	178	34·77
Total .	59	68	72	74	93	84	62	512	100·00

From a careful examination of these tables it will be seen, that the proportion of males to females born alive is as 52·42 to 47·58, and the proportion of males to females still-born is as 65·23 to 34·77.

By comparing the two tables, it will also be evident that there is an excess of 13 per 100 in the males *still-born* as contrasted with the males born *alive*, and a corresponding defect of 13 per cent. in the females *still-born*, as compared with the females born *alive*.

It also appears that the ratio of children still-born to children born alive, is 4·73 per cent.



TABLE V.—*Showing the varieties of Labour in 512 still-born.*

Labours.	1847.	1848.	1849.	1850.	1851.	1852.	1853.	Total.	Ratio per Cent.
	1848.	1849.	1850.	1851.	1852.	1853.	1854.		
Vertex Presentation . . .	26	28	30	41	43	40	35	243	47.46
Face . . .	—	—	—	—	1	—	—	1	.19
Premature Labour . . .	5	6	11	13	18	19	12	84	18.35
Breech Presentation . . .	4	3	8	6	6	5	3	35	6.03
Foot . . .	8	7	10	3	5	7	4	44	8.59
Knee . . .	1	—	—	—	—	—	—	1	.19
Shoulder . . .	—	2	1	—	—	3	—	6	1.17
Arm . . .	2	2	1	2	4	—	3	14	2.73
„ with Funis . . .	1	—	—	—	—	—	—	1	.19
Funis Presentation . . .	—	3	3	5	5	2	1	19	3.71
Placenta . . .	1	1	2	2	1	1	1	9	1.75
Delivered by Forceps . . .	1	2	1	—	1	—	—	5	.90
„ Craniotomy . . .	3	5	2	2	1	3	3	19	3.71
Flooding before Delivery . . .	—	2	—	—	5	1	—	8	1.56
Puerperal Convulsions . . .	—	1	—	—	—	—	—	1	.19
Twins . . .	2	6	2	—	1	1	—	12	2.34
Triplets . . .	4	—	—	—	2	—	—	6	1.17
Monsters . . .	1	—	—	—	—	2	—	3	.58
Cæsarian section . . .	—	—	1	—	—	—	—	1	.19
Total . . .	59	68	72	74	93	84	62	512	100.00

TABLE VI.—*Showing the varieties of Labour in 11,224 Women attended between October, 1847, and October, 1854.*

CASES.	1847.	1848.	1849.	1850.	1851.	1852.	1853.	Total.	Ratio per Cent.
	1848.	1849.	1850.	1851.	1852.	1853.	1854.		
Natural Labour . . . . .	1103	1293	1357	1634	1652	1826	688	10553	94.02
Vertex Presentation . . .	1099	1289	1356	1624	1636	1822	1686	10512	
Face . . . . .	4	4	1	10	16	4	2	41	
Premature Labour . . . . .	16	17	14	13	13	9	16	94	0.85
Ditto Induced . . . . .	1	—	—	—	1	—	—	2	
Protracted Labour . . . . .	9	23	4	8	8	7	5	64	0.57
Ditto by action of Ergot . . .	0	11	0	0	0	0	0	11	
Ditto by action of Forceps . . .	4	7	2	3	7	2	4	29	
Ditto by action of Perforator . . .	9	5	2	5	1	5	1	24	
Preternatural Labour . . . . .	26	27	47	37	69	35	31	271	2.40
Breech Presentation . . . . .	9	10	25	16	23	15	14	117	
Foot ditto . . . . .	10	8	15	7	19	12	9	80	
Shoulder ditto . . . . .	3	2	0	0	0	0	0	5	
Arm ditto . . . . .	0	3	3	9	11	4	6	36	
Hand with Head, &c. . . . .	1	2	0	8	8	1	1	16	
Funis Presentation . . . . .	2	2	4	2	3	3	0	16	
Knee ditto . . . . .	1	0	0	0	0	0	0	1	
Complex Labour . . . . .	22	26	24	25	27	22	12	158	1.40
Twins . . . . .	10	20	11	13	21	17	0	104	
Triplets . . . . .	2	0	1	0	1	0	0	4	
Placental Presentation . . . . .	1	4	2	4	3	3	0	16	
Retained Placenta . . . . .	7	0	5	0	0	0	0	12	
Puerperal Convulsions . . . . .	3	1	2	4	0	0	0	10	
Hydrocephalus . . . . .	0	1	0	0	0	0	0	1	
Ruptd. Veins of Labium . . . . .	0	0	1	0	0	0	0	1	
Albuminuria . . . . .	0	0	0	1	0	0	1	2	
Hysterical Epilepsy . . . . .	0	0	0	0	1	0	0	1	
Proclidentia . . . . .	0	0	0	1	0	0	0	1	
Prolapsed Bladder . . . . .	0	0	0	0	1	0	0	1	
Monsters . . . . .	0	0	0	0	0	2	0	2	
Rupture of Uterus . . . . .	0	0	2	1	0	0	0	3	
Flooding Labour . . . . .	12	34	4	8	9	3	0	80	0.71
Cæsarian Section . . . . .	0	0	1	1	0	0	0	2	0.01
Total . . . . .	1189	1417	1450	1725	1779	1912	1752	11224	100.00

From an analysis of the foregoing table, it appears that 10,553 labours were natural, being in the proportion of 94 per cent. Of this number, in 10,512 the vertex presented, or in the proportion of 93·6 per cent. The cases of face presentations amounted to only 41, or ·38 per cent., or 1 in 257 cases.

In the 10,512 cases of vertex presentations, 243 children were still-born, viz., in the proportion of 1 in 43, or 2·3 per cent.

### PREMATURE LABOURS.

By referring to the table, it will be seen that 96 labours are reported as premature; of this number 2 were induced, and of the 94 that occurred spontaneously, 92 were still-births, or 97·8 per cent. The ratio of premature still-births to the total number of still-births, is 18·3 per cent. Two cases of premature labour are reported as having been induced. In the first of these, craniotomy had been performed in the three previous labours, on account of an obstruction situated between the vagina and rectum, immediately beneath the promontory of the sacrum—probably ovarian. In the second case, the patient was rickety, diminutive in stature, and deformed in the pelvis, which was much contracted at the brim. Her eight previous labours were brought on prematurely; the first five at the seventh month, and the three last at the fifth month. This patient was married at twelve years of age.

The following table will show the method of operating, the length of time that elapsed from the period of induction to the commencement of pain and the completion of labour, the presentation, and event to the child.

No.	Method of Induction.	Hours before occurrence of Pain.	Hours before completion of Labour after Operation.	Presentation.	Event to Child.
1	Puncture of Membranes.	27	47	Vertex.	Dead.
2	"	36	50	"	"

## PROTRACTED LABOURS.

In 64 women the labour was protracted, being in the proportion of 0·57 per cent.

The chief causes which led to the protraction of labour were,—uterine inertia, rigidity of soft parts, disproportion between the head of the child and the pelvis, and unusual ossification of the foetal head.

In 11 cases out of 11,224, delivery was assisted by *secale cornutum*, making a proportion of about 1 in 1020 deliveries.

In 29 cases delivery was effected by the forceps : or about 1 in 387 deliveries.

It is somewhat remarkable that not a single case is reported during the septenniad, in which the *vectis* has been used, whilst in the two preceding septenniads, they nearly approached in number the *forceps* cases.

In 24 cases craniotomy was had recourse to, being in the proportion of 1 in 467·4 deliveries. If the number of women delivered by the *forceps*, be added to the number delivered by *craniotomy*, there will be 53 instrumental cases in 11,224 deliveries ; or in the proportion of 1 in 211·7.

In all the forceps cases, the long forceps was the instrument employed. Five children out of 29 delivered by forceps were stillborn. Amongst the causes which required the employment of forceps (disproportion between head and pelvis, uterine inertia, &c.), it happened in one case that the head and upper extremity presented ; in another, they were employed to expedite the delivery in a case of convulsions ; in a third, on account of severe accidental hemorrhage ; and in another they were applied to the head of the first child of a twin case, where disproportion existed between the pelvis and head : the second child being afterwards expelled by the natural powers.

The causes of *perforation* in the 24 cases in which this operation was had recourse to, were—1. Disproportion between the child's head and pelvis of mother. 2. Ossification of foetal head. 3. Puerperal convulsions. 4. A prolapsed funis (in a case where the foetus was dead). 5. Cordate pelvis. 6. Deformity, generally, and hydrocephalus, (especially marked

in the case of one woman, who had craniotomy performed in five previous labours, and delivery by forceps in three others).

In most of the cases where perforation was employed, a fair trial had been previously given to the embryoplastic instruments. In two cases sloughing of the vagina supervened, but no fistulous communication. In one of these, however, on the eve of recovery, smallpox occurred, which proved fatal.

#### PRETERNATURAL LABOURS.

The cases of preternatural labour amounted to 271, or 2·41 per cent. The number of still-born children amounted to 120 or 4·42 per cent. The nates presentations were 117, and of these deliveries 35 were still-born. The footling cases were 80, still-births, 44. The presentations of the upper extremity 41, the still-births, 20. The funis presentations amounted to 16, all of them being still-born.

#### COMPLEX LABOURS.

The number of cases of complex labour amounted to 158, or 1·40 per cent. The number of still-births amounted to 31, about 1 in 5, or 19·6 per cent. Of these 158 cases, 4 were *triplet labours*, in which 6 of the children were still-born.

#### TRIPLET CASES.

*First case.*—1st, female, living; 2d, male, still-born; 3d, male, living. All the children presented with the vertex.

*Second case.*—All still-born females. 1st, Presented with the vertex; 2d and 3d, with the nates.

*Third case.*—All males. 1st, footling, living; 2d, vertex, living, born thirty hours after first; 3d, vertex, still-born, half an hour after second.

*Fourth case.*—All females. 1st, Vertex, living; 2d, vertex, living, three hours after; 3d, hand and arm, still-born, shortly after second.



TWIN LABOURS.

These cases were 104 in number, or about 1 in 108 of the whole number of women delivered, being about 0·92 per cent. The number of twins still-born amounted to 12.

The following table will show the proportion of the sexes in twin labours, and also the nature of the presentations :

No. of Cases.	Both Males.	Both Females.	One of each Sex.
104	30	28	46

NATURE OF PRESENTATIONS.

Vertex in both . . . . .	45	Vertex and Hand . . . . .	2
Vertex and Nates . . . . .	18	Foot in both . . . . .	2
Vertex and Foot . . . . .	28	Breech and Foot . . . . .	2
Vertex and Shoulder . . . . .	1	Shoulder and Foot . . . . .	1
Vertex and Arm . . . . .	5		

In one of the cases of twin labour with the vertex of each child presenting, there was also partial placenta prævia. Of those of the vertex and foot, the second child presented the foot by the side of the head; another (first child) with the hand, the second child being born fifteen hours after. Of the vertex and arm presentations, in one case the latter came down with the head. In three, version was performed with the second child. Of the vertex and hand presentations, in one the foot also presented with the hand; and in another, the hand came down beside the head; in neither of these was version performed, but labour was completed by the natural powers.

In 16 cases "*presentation of the placenta*" occurred; in 5 of these the placenta entirely covered the os uteri, while in the remaining 11 it only partially presented. Of the former, one was a twin case, and turning was performed; this was also done in the remaining four. Of the latter, one was a *quadru-plet* presentation, in which the placenta was situated anteriorly, the head descended behind it, together with a loop of the funis and the right hand. It occurred in a woman, with a seven month's child, who had had seven miscarriages, and seven single labours, all the latter being premature except one, and

the children still-born, or dying shortly after delivery. In another case the arm and funis also presented; in a third, a foot; and in a fourth, the funis. Two deaths occurred from exhaustion, which will be afterwards noticed. Turning was performed in all these cases, except two of the partial presentations, in which, the membranes being ruptured, labour was naturally completed without further hemorrhage.

#### RETAINED PLACENTA.

Twelve cases arose from morbid adhesion, in most of which, the placenta was adherent to the anterior and upper part of the uterus. In one, death occurred from exhaustion, caused by excessive loss of blood. Fourteen cases are reported where the placenta was retained from uterine inertia, and four from irregular spasmodic contraction of the uterus. In the former, the administration of the *secale cornutum* with hand pressure effected its delivery; in the latter, it was removed by the introduction of the hand.

#### PUERPERAL CONVULSIONS.

Of this complaint there have been 10 cases, being in the proportion of 1 in 1122 cases. It is somewhat remarkable that during the last septenniad the proportion is less than half that of the previous one. During the last three years there has been no case.

CASE I.—Sophia U—, a short, stout, plethoric woman, *primipara*; had had three fits before the arrival of her attendant, and when visited she was still in convulsions. She had had œdema of face, hands, and feet, previous to the attack; but was not subject to headache or fits. She was bled, and ordered antimony with a saline, (having already taken gr. x of calomel followed by a dose of castor oil). The hair was cut off, and ice applied to the head. An enema of assafœtida was administered. She was comatose between the convulsions. The pains increased, and she gave birth to a small living female child, when she became cool and tranquil, and fell into a doze without stertor. She had no return of

the convulsions, but remained in a sleepy semi-comatose state for a day or two, when she rapidly recovered, and was quite well on the fourteenth day.

The *urine*, during the fits, was highly *albuminous*, but as she recovered, this condition gradually disappeared. An analysis of the serum of the blood, drawn during the convulsion, was made by Dr. Taylor, and *urea* was detected in it.

CASE II.—Mary Ann W—, æt. 27; second confinement. After expulsion of the head she had a well-marked epileptic convulsion, which left her in a semi-comatose state for half the day. She had no more fits after this, and did well. She had had convulsions in a previous labour. *Urine*, sp. gr. 1010, highly *albuminous*.

CASE III.—Ann L—, æt. 19; primipara, unmarried, short, stout, plethoric, and muscular; had œdema of face and legs for some time previously; no headache; a free drinker of beer and spirits. After rupture of the membranes complained of headache, and, shortly after, a convulsion came on. She was bled to sixteen ounces. The head of the child was firmly impacted at the brim of the pelvis, and there also existed obliquity of the uterus. Long forceps were applied without effect, and craniotomy was therefore performed, the fits continuing all the time at intervals. A convulsion occurred immediately after the extraction of the placenta; ten grains of calomel were given, head shorn, and cold evaporating lotions applied. Antimony and calomel were ordered, and cupping was prescribed, on account of the continuance of the fits. On the following day she was removed into the hospital in an insensible state. The puncture of venesection suppurated, and an erysipelatous blush extended up the arm. Convulsive movements of the jaw and left arm persisted for some time; she appeared to get more conscious, but eventually the erysipelas extended over the whole abdomen to the external genital organs, to the perinæum, (which was slightly lacerated), and down the thighs; diarrhœa also set in, and she sank on the morning of the fifth day. No post-mortem was allowed. The *urine* was very *albuminous* at first, but the albumen gradually decreased till the day of her death.



CASE IV.—Sarah C—, æt. 24; first confinement, a nervous irritable woman, was delivered of a living child after a lingering labour. The day following she complained of pain in her head, with constant thirst; occasional delirium, lochia offensive. Calomel and Dover's powder were administered, and an injection of turpentine. On the third morning after labour she was scarcely conscious, and often delirious; teeth clenched, mouth drawn to one side, short and hurried respiration, but no true convulsion. Sinapisms were applied to the feet; hair removed and cold applied, and salines with tartarized antimony exhibited. The coma continuing, a blister was applied to the nape of the neck, and calomel combined with camphor and hyoscyamus exhibited every three hours. She gradually became worse, and sank on the fourth day after delivery, and third from the supervention of the symptoms. Wine and opium were administered latterly, but she died at last in a convulsion. Urine was of sp. gr. 1010, highly *albuminous*.

*Sectio cadaveris*.—Veins of dura mater turgid; serous effusion beneath arachnoid and base of brain; no abnormal effusion in the ventricles; substance of brain healthy. An unusual quantity of fluid in the pericardium; heart healthy. Abnormal effusion into the peritoneum, with shreds of false membrane in neighbourhood of uterus: no pus in the uterine sinuses. Fragments of the deciduous membrane were being detached from the interior of the uterus.

CASE V.—Caroline E—, æt. 43; third confinement; married a second time; a thin delicate woman; had an interval of ten years between her second and present confinement; four years a widow. Has suffered frequently from headache and œdema of the lower extremities during the latter months of gestation; never had convulsions previously. Present labour natural; living female child. Sixteen hours after delivery she had a fit. Four grains of calomel, followed by a dose of castor oil, were exhibited; salines every four hours, and cold was applied to the head. Another fit occurred, and she vomited. A soap enema was administered, and the mixture repeated, with  $\mathfrak{mxx}$  of Vin. Ant. Tart. Under this treatment the symptoms gradually disappeared, and with the exception of an attack of diarrhœa, which was checked by the exhibition



of chalk and opium, she gradually recovered, and on the eleventh day was convalescent. The *urine* was *albuminous*.

CASE VI.—Sarah B—, æt. 18; primipara, a stout fair-complexioned young woman, of strong muscular development. When summoned to her, her attendant found her perfectly unconscious, breathing stertorously, and the respiration mixed with mucous and bronchial râles; dilated pupil; the face and lower extremities œdematous, pulse 110, very compressible. She shortly afterwards had an epileptic fit, when she was bled to six ounces, her head shorn, cold applied, and an ounce of castor oil given directly. The venesection lowered the pulse, but the fits recurred every ten minutes. Cal., gr. x, given immediately, followed by a turpentine enema, and tartarized antimony with salines, ice to the head, &c. As labour did not seem to be at all progressive, the os being very little dilated, the membranes were artificially ruptured, and upon the descent of the head to the outlet, forceps were applied, and a still-born female child delivered, whose limbs were tetanic and rigid, as if it participated in the convulsions of its mother. Two severe fits followed the delivery of the child. They continued at intervals for some time, attended with active delirium. All the symptoms were evidently increasing, and she gradually sank, and died about forty-three hours after the first attack of convulsions. She was never conscious after the first fit. *Urine* densely *albuminous*, sp. gr. 1028.

*Sectio cadaveris, twenty hours after death.*—Dura mater somewhat adherent; brain-surface much injected, softening of the posterior part of the cerebrum, no ventricular effusion; kidneys coarse and congested, capsules adherent.

CASE VII.—Elizabeth F—, æt. 50; eleventh confinement. Has enjoyed ordinary good health, but has been the subject of severe fits, which have come on after long intervals, attended with protracted insensibility. Thirteen years ago she suffered from drowsiness and a sense of fulness in the head, for which she was bled, with great relief. When visited, she was comatose, with convulsions at intervals. Calomel and antimony were administered, with sinapisms to the legs. The forceps were applied, and a living female child extracted, with little

difficulty. The coma remained the same after delivery, but the convulsions abated; she sank, however, on the fourth day. *Urine* densely *albuminous*.

*Sectio cadaveris*.—Old and recent effusion beneath arachnoid; substance of brain slightly ecchymosed in places, the rest healthy; unusual quantity of fluid in ventricles; abdominal and thoracic viscera healthy.

CASE VIII.—Margaret D—, æt. 28; first confinement, of fair complexion, short stature, and plethoric; married. Had always enjoyed good health, but was excitable. An aunt died some short time before, undelivered, with puerperal convulsions; and her father was the subject of albuminuria. She had slight œdema about the face and hands previously to labour, and dreaded her approaching confinement; she was also suffering from bronchitis. She was attacked with puerperal convulsions in the second stage of labour; the pulse was about 40 and labouring during the fit, and 120 in the interval. Two other fits occurred, previous to the delivery of a living female child by the short forceps, after which she became more tranquil. Two fits occurred within an hour after delivery, and she was violently delirious. Twelve ounces of blood were taken from the arm; the head shorn, and cold applied; and a powder, composed of calomel, jalap, and tartarized antimony, administered. The fits recurred every twenty minutes; and they subsequently became completely tetanic, producing opisthotonos, &c. Blisters were applied to the nape of the neck, sinapisms to the calves of the legs and soles of the feet, and antimony and salines were given. The last fit occurred on the evening of the second day after confinement, making the fortieth (four happening before delivery, thirty-six after). She remained comatose for some hours after this last convulsion, when the symptoms abated; she was then attacked with more severe symptoms of bronchitis, which proved fatal. The *urine* was densely *albuminous* at first, but gradually became less so till her death. No post-mortem examination allowed.

CASE IX.—This case was one of labour, complicated with epileptic fits, and followed by peritonitis. The subject of it afterwards recovered.

CASE X.—The report states that in this case, one of puerperal convulsions, the woman was instrumentally delivered.

It will thus be seen that, of the nine cases of convulsions reported, five proved fatal to the mothers; and out of the eight fully reported cases, two children were still-born and six alive. Three of the children were delivered by the forceps, and one by the perforator. Five cases were first labours (only one being reported as illegitimate); one was a second labour, in which the woman had had convulsions in her previous labour; one was a third labour, in which for the first time convulsions occurred; and one was the eleventh confinement: this occurred in a woman who had been subject to epileptic fits for nearly thirteen years, and in whom old-standing disease of the membranes of the brain was found, in addition to the recent affection.

In all the cases the *urine* was found *albuminous*.

#### HYDROCEPHALUS.

These cases are only two in number; one of which is mentioned in the table, under the head of protracted labour, as delivered by perforator.

CASE I.—Mary P—, æt. 24; fourth confinement, previous labours good; had had very good health during her pregnancy. Labour commenced at 10 a.m.; she was first seen at 1 p.m., when the os uteri was found about the size of a shilling, soft and dilatable, and the pains recurring every five minutes. At 9 p.m., the os being fully dilated, the membranes were ruptured, and the head partly entered the brim, but, notwithstanding the most violent expulsive pains, it remained fixed in that position. The death of the fœtus was determined by the silence of the heart's beats, and then craniotomy was had recourse to; a large quantity of fluid was let out, and delivery speedily completed. The abdomen was tympanitic and very tender, pulse 100, skin hot. Ordered Tr. Opii, ʒss. She slept very little during that night, and in the morning complained of considerable pain in the abdomen, which still



remained tympanitic and tender; she had also a dry hacking cough. Having passed no urine since the labour, a pint and a half of very dark-coloured water was drawn off by the catheter. Pulse 106, soft and compressible; tongue clean and moist; lochia scanty. Some Dover's powder, with calomel, and a cough mixture, were ordered, and hot fomentations to the abdomen; and in two days she was considerably better. From this time she continued to improve, and in four days was convalescent. The distension and pain, principally of the left side of the abdomen, continued, however, to trouble her for some little time after the other symptoms had disappeared.

The foetal head, when examined after delivery, was found to be very large, and, after the brain was removed, would contain one pint and three quarters of fluid, and measured, while distended, seventeen inches and a half in circumference.

CASE II.—Hannah F—, a married Irishwoman, æt. 29; eighth confinement. When summoned, at 6 p.m., the attendant found the os uteri partially dilated, the membranes ruptured, and the pains powerful and quick, with the head presenting. After two hours, finding that no progress was made and seeing nothing to apprehend, he left the patient, and on his return, in two hours afterwards, found that the funis had descended by the side of the head, and could not be returned. Assistance having been procured from the Lying-in Charity, and the cord having ceased to pulsate, labour was completed by craniotomy, and the head was found to be hydrocephalic, measuring in its collapsed state sixteen inches in circumference. The patient recovered favorably.

#### PUERPERAL EPILEPSY.

A fatal case of this nature is reported, as happening during pregnancy, of which the particulars are as follows:

Margaret B—, æt. 21, of Irish parents, though born in England, is of middle stature, pregnant with first child. She has on one or two other occasions suffered from "*fits*," the nature of which is indefinite. At 4 p.m. she complained of headache, vomited, and became also very restless. About

10 o'clock fell from her bed to the floor insensible, soon however became conscious, but upon being raised relapsed into a state of perfect unconsciousness, from which she never recovered; her face became distorted; she was motionless and comatose, with great general congestion; her teeth were clenched, and she foamed at the mouth. The pupils were dilated and insensible to light; pulse rapid, but feeble; respiration slow, but not stertorous; she passed her urine involuntarily.

On vaginal examination it was found that labour had not commenced (she being supposed to be about eight months gone in pregnancy). The attendant endeavoured to revive her, but she died very shortly after his arrival.

*Sectio cadaveris, twenty-four hours after death.*—Aspect of body generally anæmic; the uterus contained a fully developed male child, with the head presenting; the abdominal and thoracic viscera were healthy, though congested; the integuments of the cranium and the vessels of the brain were congested, and the substance of the brain, when cut into, was freely scattered with bleeding blood-vessels; the arachnoid was thickened.

#### LABOUR IN A PARALYTIC WOMAN.

A case of natural labour is reported as having occurred in a woman affected with paralysis, which had supervened during the second month of this her sixth pregnancy, accompanied with numbness and loss of power in her limbs and sides, more particularly in the right. At the time of her labour she had perfect hemiplegia, the symptoms having gradually increased up to this time; her countenance was blank, without expression; voice muffled; left pupil much dilated; tongue protruded to right side; no loss of power over the sphincters. She had a perfectly easy labour, and was delivered of a living male child.

Labour was effected by the uterus alone, which acted powerfully, the abdominal muscles being inactive throughout.



## RUPTURED VARICOSE VEIN OF LABIUM—DEATH.

Catherine C—, æt. 31, married, and the mother of five children ; of middle stature, with dark hair and sallow complexion. On September 5th, 1850, her attendant was summoned, and at 9 a.m. he found her in a state of collapse from hemorrhage. Her dress and bed-clothes were saturated with blood, which was also running upon the floor. Brandy was immediately administered, but she sank, and died in about three quarters of an hour ; no hemorrhage in the meanwhile having occurred. On inquiry it was found, that, whilst fighting with her husband, she suddenly exclaimed that the child was being born. A friend immediately examined her, and found a tumour, apparently external to the vagina, which, as soon as she was lifted upon the bed, burst, and produced this fatal hemorrhage.

*Sectio cadaveris, seventy-four hours after death.*—Body much decomposed ; a bruise existed under the right eye ; the vagina was free from blood ; os uteri high up, and undilated. The uterus was found to contain a seventh or eighth month male foetus ; the placenta was attached to the fundus ; and the membranes were entire. A corpus luteum was found in the right ovary. Liver soft and coarse ; kidneys pale. There existed old pleuritic adhesions in the thorax. Heart empty ; brain pale. The cause of death was the rupture of a varicose vein in the right labium, which produced a large thrombus in the areolar tissue, capable of holding ten ounces of blood ; this, suddenly bursting, had caused the fatal syncope. The inner surface of the labium was torn to the extent of an inch.

One case of “ *Ovarian Tumour obstructing Labour,*” till the operation of puncture was performed, is reported. The puncture was made per rectum, and about eight ounces of a thick fluid was discharged. After which the labour proceeded naturally, but the child (a male) was still-born.

## RUPTURE OF UTERUS.

Three cases of ruptured uterus are reported during the seven years embraced in this report. They hold a proportion to the other cases as one in 3741. They occurred in the years 1849-51, and two happened within a month of each other. They are as follows:

CASE I.—Sarah B—, æt. 28, married six years; had had three children, of which one was alive; had never miscarried. All her previous labours had been lingering, especially the last. She had lately followed the occupation of a sempstress, and had been in poor circumstances. For the last two months of pregnancy she had complained of a severe dragging pain in the abdomen and back, and her diet during this period had been scanty. At 2.45 a.m., on the 16th of November, the attendant was summoned, and found her lying upon a bed suffering from severe expulsive labour pains. On inquiry he found that the membranes had ruptured half an hour previously, and that these severe pains had commenced at 1 o'clock. On examination the os uteri was found fully dilated, and the head presenting at the upper part of the vagina, looking towards the sacrum. In half an hour it entered into the cavity in the first position. The pains came on at intervals of every two or three minutes, and there seemed at first a probability of a speedy termination to the labour. A *caput succedaneum* began to form, so that the head could be felt barely within half an inch of the external parts, which had now become dilated. From this time the head did not advance, but remained firmly impacted in the cavity; the pains, though strong and expulsive, having no influence upon it. About 7 o'clock a.m. the patient rather suddenly complained of a severe burning pain in the abdomen, extending to the chest, which was succeeded by vomiting, and signs of exhaustion; the presenting part at the same time receding. The child was delivered by forceps, and in half an hour afterwards the woman expired.

*Sectio cadaveris, twenty-four hours after death.*—Surface of

body pale; mammae well developed; abdomen distended; purpuraceous spots in front of both legs; abdominal parietes coated with fat, but muscles thin. On opening the peritoneum the cavity was found filled with about half a gallon of blood and clots. The uterus was contracted and lacerated. A rent was seen commencing towards the left side of the posterior wall of the vagina, immediately beneath the os uteri, running at first almost transversely across the vagina, separating it from the uterus, then obliquely upwards to the right side through the walls of the uterus for two or three inches, and continuing on to the right broad ligament; the whole rent being nearly seven inches in extent. The right ovary contained a well-formed corpus luteum; the left was small and atrophied. As regards the bones, the promontory of the sacrum was sharp and prominent, and the tuberosities of the ischia were too near each other. The bladder was empty; liver and spleen small. Milk-white spots were seen under the investing tunic of the kidney in the secreting tissue, and both were very large. No other parts were examined.

CASE II.—Julia S—, æt. 38, married; was taken in labour with her thirteenth child, December 20th. Most of her previous labours had been tedious (one instrumental). She was of short stature, average stoutness, and had generally enjoyed good health, but was nervous and irritable.

During the last three months of pregnancy she had complained of a sharp pain in the abdomen to the right of the umbilicus, and six weeks before, she received a shock from being told that her husband had fallen into the river, and also from her children being attacked with cholera. The attendant was summoned to her at 11 p.m. The membranes were ruptured; pains irregular and at long intervals; and labour went on slowly until 10.30 p.m. the following day, when signs of collapse set in suddenly, and she expired before further assistance could be obtained or delivery completed.

*Sectio cadaveris, sixteen hours after death.*—On opening the abdomen old marks of peritonitis were found. A small quantity of serum existed in the cavity of the peritoneum, and about a pint of blood was found effused between the layers of the



right broad ligament. On carefully removing the uterus and its contents a laceration was found, four inches in length, extending from the inferior and right side of the uterus to the broad ligament. Around the laceration the tissue of the uterus was soft, and the peritoneum covering the organ was much congested. The child was a male, and rather large.

CASE III.—Hannah W—, æt. 36; was taken in labour at 3 p.m. on July 9th, and first seen by her attendant at 6.30 p.m. The membranes had ruptured, and the head presented, the os uteri being dilated to the size of a crown-piece, and soft parts relaxed. The pains continued with little interruption till 2 a.m. the following morning, and the head gradually progressed. Hemorrhage took place about this time, and the pains became less frequent. At 3 a.m., after a severe pain, her countenance became very pale, pulse small, and she complained of faintness and pain in her stomach, with dyspnœa. The pains ceased, and brandy was immediately administered. Assistance from the Lying-in Charity was sent for, but before the arrival of the resident obstetrician, the patient had expired undelivered, the hemorrhage having continued in the meanwhile.

*Sectio cadaveris, twenty-eight hours after death.*—Body plump; no indications externally of pelvic deformity. Blood had been trickling from the vagina after death. On opening the abdomen a quantity of blood was found extravasated into the peritoneal cavity, from a rent in the posterior wall of the uterus, low down, about three inches in length. Other parts of the body not examined.

#### CASES OF "SUPPOSED RUPTURE OF THE UTERUS."

Three cases of "supposed rupture of the uterus" have occurred, two of which proved fatal, the third recovered; in the two fatal cases no autopsy was permitted, therefore the suspicions which had been excited by the symptoms before death could not be proved by post-mortem examination.

CASE 1.—Eleanor S—, æt. 36, an Irishwoman, of dark complexion, married; was taken in labour with her sixth child, April 12th, at 11 p.m. When visited, the os uteri was dilated to the size of a crown-piece, pains regular (every five minutes), head presenting. At 12 the os uteri was fully dilated, and the membranes ruptured. The pains increased till 5 a.m. the following morning, and then gradually subsided, and ceased between 7 and 8. Vomiting, abdominal tenderness, a rapid, feeble pulse, coldness of the face and extremities, and hurried breathing indicated considerable depression, which soon became more urgent. The forceps were now attempted to be applied, but the head receded. A little blood escaped per vaginam. Turning was now had recourse to, and the feet brought down, but there was some difficulty in delivering the head. The placenta came away without hemorrhage, and the uterus contracted well. Tr. Opii, ʒj, was administered, with brandy and water. In an hour the pulse began to increase in volume, and the patient seemed more comfortable, although she still complained of abdominal tenderness on the slightest pressure. In the evening the pulse became more full and slow, and the abdomen was still tender and tympanitic, but the breathing was less hurried. Tr. Opii administered. On the following day she had slept a little, had no lochial discharge, and was ordered Hydr. Chlor., gr. ij; Pulv. Opii, gr. j, 4tis horis. On the 15th she had a great deal of febrile reaction, for which saline effervescent mixture with ammonia was prescribed, in addition to the other medicines, and an enema. Lochial discharge slight, no milk. On the 16th the enema had not operated; uterus very large, reaching to umbilicus, and tender. No action of bowels. Enema Hord. cum Ol. Ricini. Lochia ceased. The following day had vomiting and purging, with increased abdominal tenderness. Rep. Epith. Terebinth. 19th. Pain in the abdomen severe, uterus enlarged and sensitive. Lin. Hydr. Abd. infric. et Opii, gr. j, ter die. The next day she was better generally, and was ordered four ounces of wine, with a chop. 22d. Purging commenced again with slight vomiting, after partaking of beef tea. On the 24th the purging and vomiting had ceased. Uterus smaller and less sensitive; she was very weak. To take effervescent mixture, with Ammon. Sesquicarb., gr. iv, ter die. From this time she continued to



improve, though having occasional attacks of purging and vomiting, which were checked by chalk and opium, &c.; and by the 10th of the following month she was convalescent.

CASE II.—Mary H—, æt. 43, of middle stature, dark, and sallow complexion; had always been temperate and enjoyed good health; was the mother of ten children. The first seven labours were good, the last three tedious, and one was instrumental. During the night of the 14th of April, she passed a large quantity of blood, which continued to drain from her, but without labour pains, till the 17th, at 6 p.m., when her attendant was summoned, who found the os uteri almost fully dilated, and the head at the brim. Her pains were then regular, and although sharp, inefficient. At 5 a.m. the labour had not at all progressed, her pulse was weak, the pains had subsided, and the external parts œdematous. Ammonia was immediately administered, and she was then left for an hour and a half. When her attendant was again sent for, he found her quite collapsed and the pulse scarcely perceptible. Ammonia was repeated, and further assistance procured from the Lying-in Charity. On examination, the head was felt still in the same position at the brim, and the external parts much swollen; she complained also of a sensation of fulness about the body. The obstetric physician regarded the case as one of rupture of the uterus. After the administration of stimulants, craniotomy was performed; but before delivery by the crotchet could be completed, the symptoms of exhaustion became so urgent that turning was had recourse to. The pulse, which during the operation began to get weak, now became still weaker, and, in spite of stimulants, &c., the patient expired, about three quarters of an hour after delivery. No autopsy was permitted.

CASE III.—Hannah M—, æt. 30, short, somewhat stout, of dark complexion; second confinement. Sent for her attendant at 7 p.m., November 6th, 1853. Labour pains had come on some hours previously; the membranes had ruptured in the afternoon, and the feet presented. The pains, previously regular, had now ceased; the os was dilated to the size of half a

crown and dilatable; the soft parts were cool, and the powers good. She had vomited during the night. The breech now presented. Pains continued at intervals during the day, and a few clots of blood passed. At 7 p.m. (7th) the pains increased, and labour went on until 8 a.m., when the pains suddenly ceased, and urgent vomiting, faintness, and a sensation of coldness supervened. At 9½ a.m. she was pulseless, surface cold, countenance sunken and anxious. Delivery was hastened, and a dead female child withdrawn. The placenta soon followed, and but little blood was subsequently lost. She did not rally after this, but died at 7 p.m.

No post-mortem examination of the body was allowed.

#### FLOODING LABOUR.

The number of cases of flooding labour amounts to 80, or 0·7 per cent. Under this division are included 23 cases of accidental hemorrhage, 15 cases of hemorrhage after the birth of the child and before the expulsion of the placenta, and 42 after the expulsion of the placenta. In 3 cases out of the 23, the hemorrhage (accidental) proved fatal, the placenta being found, post mortem, to be detached for nearly its whole extent; the uterus containing a large quantity of blood. In one case the child was premature, and presented by the breech. No blood in these cases showed itself externally, and the deaths were very sudden.<sup>1</sup>

#### MONSTERS.

Though two only are mentioned in the table, on carefully looking through the cases there is mention made of two others, making in all four. In one case, the legs were joined together, but the feet distinct; and the sexual organs very imperfectly indicated.

The three remaining children were anencephalous; one of them (a still-born male) was premature, at the seventh month;

<sup>1</sup> Two of these cases are related by Dr. Oldham, in his paper on "Internal Uterine Hemorrhage," p. 94.

the two others were, respectively, a still-born male and female, fully developed.

### CÆSARIAN SECTION.

Two cases requiring this most formidable operation are reported. In one case, both mother and child were saved; in the other, both died.

They happened in the years 1850 and 1851, and are related in detail, with clinical remarks, in 'Guy's Reports,' series ii, vol. vii, p. 426; and 'Medico-Chirurgical Transactions,' vol. xxxiv.

### FATAL CASES.

The number of fatal cases during the seven years is 65, or about 1 in 172 cases.

*Table of 65 Fatal Cases.*

Disease.	No. of Cases.
Peritonitis, Metro-phlebitis, Puerperal fever . . . . .	35
Bronchitis, Pleurisy, Pneumonia . . . . .	3
Convulsions . . . . .	6
Labial thrombus . . . . .	1
Jaundice . . . . .	1
Placenta prævia . . . . .	3
Laceration of cervix . . . . .	1
Hemorrhage . . . . .	5
Ruptured uterus . . . . .	5
Cholera . . . . .	2
Phthisis . . . . .	1
Cæsarian section . . . . .	1
Exhaustion after delivery . . . . .	1
	<hr/> 65

The following is a short abstract of the cases, taken as they appear in the books of the Charity :

CASE I.—Age 24; first confinement; presentation, vertex; labour, natural; period of attack, second day after delivery;

disease, *peritonitis*; fatal on the seventh day. Treatment: leeches, calomel, opium, fomentations, wine, cinchona.

CASE II.—Age 44; ninth confinement; presentation, vertex; delivery, by forceps; death immediately after delivery, from *hemorrhage*; sudden exhaustion. On post-mortem examination, four pints of uncoagulated blood found in peritoneal cavity; no laceration of uterus, but a vessel in the broad ligament had given way; uterine walls remarkably thin; kidneys granular.

CASE III.—Age 30; fourth confinement; presentation, vertex; natural labour; period of attack, third day; disease, *metro-phlebitis* and *pleurisy*; death in thirty-six hours. Treatment: fomentations, calomel, antimony, opium, blisters.

CASE IV.—Age 24; first confinement; presentation, vertex; natural labour; attack, second day; disease, *convulsions*; fatal on the fourth day. Treatment: calomel, Ant. Tart., blisters, lotion.

CASE V.—Age 27; third confinement; presentation, vertex; labour, natural; period of attack, fifteen days after delivery; disease, *phlebitis* and *pleuro-pneumonia*; death on eighteenth day. Treatment: calomel, opium, leeches, blisters, wine, ammonia.

CASE VI.—Age 20; first confinement; presentation, vertex; attack, on third day; disease, *influenza*, *peritonitis*; death on sixth day. Treatment: leeches, calomel, opium.

CASE VII.—Age 33; fourth confinement; presentation, vertex; natural; period of attack, third day; disease, *peritonitis*; fatal on the fourth day. Treatment: leeches, castor oil, ammonia, &c.

CASE VIII.—Age not stated; second confinement; breech presentation; attack, on second day; disease, *peritonitis*; fatal, fourth day. Treatment: fomentations, calomel and opium, &c.

CASE IX.—Age 35; seventh confinement; presentation, vertex; labour, natural; period of attack, the day after delivery; disease, *peritonitis*; fatal, third day. Treatment: leeches, fomentations, calomel, and opium.

CASE X.—Age 30; ninth confinement; vertex presentation; lingering labour; attack, on second day; disease, *bronchitis*,



*pleuro-pneumonia* ; fatal on the sixteenth day. Treatment : calomel, opium, blisters, wine, brandy. On post-mortem examination, an abscess was discovered between the *liver* and *diaphragm*, with right pleuritic effusion ; universal bronchitis.

CASE XI.—Age 34 ; first confinement ; natural labour ; vertex presentation ; attack, on sixth day ; disease, *peritonitis* ; fatal, ninth day. Treatment : calomel, antimony, opium, sinapisms.

CASE XII.—Age 30 ; eighth confinement ; labour, natural ; presentation, vertex ; period of attack, fourth day ; disease, *metro-phlebitis*. Treatment : calomel, opium, fomentations ; fatal on the twelfth day. On post-mortem examination, pus found in uterine veins.

CASE XIII.—Age 34 ; seventh confinement ; vertex presentation ; natural labour ; attack, fourth day ; disease, *diffused phlebitis* ; fatal, seventh day. Treatment : calomel, opium, ammonia, sinapisms. On post-mortem examination, was found, *peritoneal*, *pleuritic*, and *pericardiac* effusion, with pus in pelvis ; metritis.

CASE XIV.—Age 22 ; first confinement ; vertex presentation ; post-partum hemorrhage ; attacked the day after delivery with *peritonitis* ; fatal, third day. Treatment : calomel, opium, fomentations.

CASE XV.—Age 42 ; fifteenth confinement ; lingering labour, complex, twins ; first presented by the head, second by the feet ; attacked shortly after delivery with *puerperal fever* ; fatal on the sixth day. Treatment : leeches, calomel, opium, ammonia, brandy. On post-mortem examination, pus found in uterine sinuses, ovaries, and liver ; also in cellular tissue of pelvis ; peritoneal effusion. Both children died in four days from convulsions.

CASE XVI.—Age 21 ; first confinement ; vertex presentation ; attacked on third day by *puerperal fever* ; fatal on eighth day. Treatment : leeches, calomel, opium, fomentations, ammonia, brandy. On post-mortem examination, pus found in uterine sinuses.

CASE XVII.—Age 30 ; third confinement ; natural presentation ; death in about fifteen minutes after delivery, apparently from *exhaustion*. No post-mortem.



CASE XVIII.—Age 32 ; third confinement ; natural presentation ; attacked on the third day by *puerperal fever* ; fatal on the third day. Treatment : calomel, opium, ammonia, &c.

CASE XIX.—Age 29 ; third confinement ; vertex presentation ; natural labour ; attacked on fifth day with *puerperal peritonitis*, fatal ninth day. Treatment : calomel, opium, ammonia, brandy. No pus in uterine veins.

CASE XX.—Age 28 ; fourth confinement ; twins, both vertex presentations ; attacked twenty-four hours after delivery with *puerperal fever* ; fatal on the third day. Treatment : leeches, calomel, opium, brandy, ammonia. N.B. Two days after the death of the mother, the male twin died of erysipelas, and on the following day the female died of convulsions.

CASE XXI.—Age 26 ; fifth confinement ; natural presentation ; attacked the second day with *metro-phlebitis* ; fatal fifth day. Treatment : Dover's powder, calomel, opium, leeches. Pus found in the uterine sinuses.

CASE XXII.—Age 19 ; first confinement ; *convulsions* before delivery ; delivered by perforator ; died on the fifth day after the operation. Treatment : venesection, calomel, antimony, hyoscyamus, evaporating lotion and ice to head, &c.

CASE XXIII.—Age 30 ; second confinement ; natural presentation ; seized the second day with *puerperal fever* ; fatal on fourth day. Treatment : calomel, Dover's powder, leeches, mercurial inunction, wine, &c. Child died three days after from *erysipelas*.

CASE XXIV.—Age 30 ; fourth confinement ; natural labour ; post-partum hemorrhage ; attacked fifth day with *metro-phlebitis* ; fatal eighth day. Treatment : leeches, calomel, opium, &c.

CASE XXV.—Age 38 ; seventh confinement ; vertex presentation ; placenta retained from hour-glass contraction ; hemorrhage ; attacked the third day with *puerperal fever* ; fatal seventh day. Treatment : uterine injections, fomentations, calomel, opium, mercurial inunction.

CASE XXVI.—Age 38 ; eighth confinement ; twins ; both vertex presentations ; third day attacked with *puerperal fever* ; fatal sixth day. Treatment : calomel, opium, vaginal injections, ammonia, brandy. Pus found in uterine veins ; lobular pneumonia.

CASE XXVII.—Age 41; eleventh confinement; partial placental presentation; version; death from *exhaustion* six days after delivery. Stimulants, opium.

CASE XXVIII.—Age 22; first confinement; natural presentation; twelfth day after delivery seized with *pneumonia* and *phlegmasia dolens*; fatal twenty-sixth day. Treatment: Hydrargyrum cum Cretâ, Dover's powder, leeches, fomentations, ammonia, brandy. On post-mortem examination, double pneumonia; femoral and inguinal veins impervious; an abscess existed in the cellular tissue of right side of pelvis.

CASE XXIX.—Age 36; eighth confinement; natural labour; seized with *cholera* shortly after delivery; fatal in forty-four hours. Treatment: chalk and opium, brandy, &c.

CASE XXX.—Age 19; first confinement; vertex presentation; partial adherent placenta; attacked on the fourth day with *puerperal fever*; fatal on sixteenth day. Treatment: calomel, opium, ammonia. Was unmarried; neglected greatly by her friends and relations.

CASE XXXI.—Age 28; fourth confinement; vertex presentation; delivered by forceps; died half an hour after delivery, from *ruptured uterus*. (*Vide Report.*)

CASE XXXII.—Age 38; thirteenth confinement; vertex presentation; died undelivered from *ruptured uterus*. (*Vide Report.*)

CASE XXXIII.—Age 24; third confinement; natural labour; attacked on the fifth day with *pleurisy*; fatal sixteenth day. Treatment: calomel, Antim. Tart., opium, ammonia, blisters.

CASE XXXIV.—Age 30; sixth confinement; foot presentation; difficulty in delivering the head; on the second day *metro-phlebitis*, which proved fatal on the sixth day. Treatment: fomentations, calomel, Ant. Tart., opium.

CASE XXXV.—Age 21; first confinement; died previous to labour from *puerperal epilepsy*. (*Vide Report.*)

CASE XXXVI.—Age 18; first confinement; vertex presentation; convulsions previous to labour; delivered by forceps; died twelve hours after first attack, and eight after delivery. Treatment: venesection, calomel, Antim. Tart. (*Vide Report.*)

CASE XXXVII.—Age 31; sixth confinement; died during pregnancy from labial *thrombus*. (*Vide Report.*)

CASE XXXVIII.—Died after operation of *Cæsarian section*. (*Vide Report*.)

CASE XXXIX.—Age 40; tenth confinement; vertex presentation; woman died undelivered from *intra-uterine hemorrhage*.

CASE XL.—Age 36; sixth confinement; vertex presentation; natural labour; nine hours after delivery, attacked by *jaundice*, which proved fatal on the fourth day. Treatment: purgatives, quinine, sulphuric ether, stimulants. Child died half an hour post partum; *jaundiced*.

CASE XLI.—Age 40; ninth confinement; natural labour; third day attacked with *puerperal fever*, fatal on the sixth day. Treatment: fomentations, calomel, opium, Ant. Tart., ammonia.

CASE XLII.—Age 28; first confinement; natural labour; attacked on the fourth day with *puerperal fever*, which proved fatal on the ninth. Treatment: calomel, opium, ammonia, &c.

CASE XLIII.—Age 43; eleventh confinement; vertex presentation; delivered by forceps; *convulsions*, fatal four days after delivery. Treatment: bleeding, cupping on temples, calomel, antimony, cold, ammonia. (*Vide Report*.)

CASE XLIV.—Age 25; fourth confinement; natural presentation; attacked on the fourth day with *puerperal fever*, fatal on the seventh day. Treatment: Dover's powder, Hydr. c. Cretâ, ammonia.

CASE XLV.—Age 28; first confinement; vertex presentation; *convulsions* prior to delivery, which latter was effected by forceps; died from *bronchitis* on the sixth day after delivery. Treatment: venesection, calomel, opium, blisters. (*Vide Report*.)

CASE XLVI.—Age 36; sixth confinement; breech presentation; third day after delivery was seized with *puerperal fever*, died in twenty-four hours. Treatment: poppy fomentations, calomel, opium, wine, &c. Pus in uterine sinuses and about pelvis.

CASE XLVII.—Age 36; first confinement; vertex presentation; died undelivered, from *ruptured uterus*. (*Vide Report*.)

CASE XLVIII.—Age 38; tenth confinement; *placenta prævia*; version; death from *exhaustion*, about fifteen minutes after delivery.

CASE XLIX.—Age 20; first confinement; natural labour; on the second day was attacked with *peritonitis*, which proved fatal on the fourth day. Treatment: calomel, Ant. Tart., opium, wine, brandy, &c.



CASE L.—Age not stated; death from *internal hemorrhage*; undelivered. On post-mortem examination, placenta found to be almost entirely detached; a seven-months child found in uterus, presenting by the breech.

CASE LI.—Age 18; first confinement; vertex presentation; delivered by forceps; *ante-partum hemorrhage*; died from *exhaustion*, eleven days after delivery. Treatment: wine, opium, plug, &c.

CASE LII.—Age 37; fifth confinement; natural presentation; attacked on the fifth day with *phlebitis* and *phlegmasia dolens*, fatal on the tenth day. Treatment: leeches, opium, brandy, &c. On post-mortem examination, pus was found in uterine sinuses; also an abscess between the layers of broad ligament.

CASE LIII.—Age 36; eighth confinement; natural labour; twenty-four hours after delivery was seized with *puerperal fever*, fatal on the fourth day. Treatment: fomentations, poultices, opium, ammonia. Pus found in Fallopian tubes and cavity of uterus.

CASE LIV.—Age 43; eleventh confinement; died three quarters of an hour after delivery, from *rupture of the uterus*. Perforation was first had recourse to, and subsequently turning.

CASE LV.—Age 35; sixth confinement; partial *placenta prævia*; vertex presentation; version; fatal on the ninth day after delivery, from exhaustion. Treatment: brandy, opium, &c.

CASE LVI.—Age 42; ninth confinement; died of *phlebitis* following *abortion*, in which the placenta could not be removed. (No distinct report.)

CASE LVII.—Age 30; third confinement; natural presentation; attacked on the third day with *uterine phlebitis*, fatal on the tenth day. Treatment: Dover's powder, opium, ammonia, &c.

CASE LVIII.—Age 22; first confinement; vertex presentation; craniotomy; attacked, twenty-four hours after delivery, with *phlebitis* and *sloughing vagina*; died from exhaustion on the tenth day. Treatment: opium, wine, Lotio Acid. Nitr., yeast poultices, &c.

CASE LIX.—Age 30; second confinement; footling presentation; died, nine hours after delivery, from *supposed rupture of the uterus*. (*Vide Report*.)

CASE LX.—Age 34; seventh confinement; hand presentation; version; hemorrhage before the birth of the placenta, which was adherent; fatal twenty-four hours after delivery. On post-mortem examination, there was found *laceration of the cervix uteri*.

CASE LXI.—Age 40; fifth confinement; vertex presentation; delivery by forceps; died from *peritonitis* on the eighth day.

CASE LXII.—Died from *phthisis*. (No distinct report.)

CASE LXIII.—Age 31; died from *hemorrhage*; a firmly adherent placenta; natural labour; seventh confinement.

CASE LXIV.—Death from *choleraic diarrhæa*. (No report.)

CASE LXV.—Death from *peritonitis*. (No clear report.)



## PART II.

### REPORT OF THE LYING-IN CHARITY FOR TWENTY-ONE YEARS.

From October, 1833, to October, 1854, 22,498 women have been attended by the pupils of Guy's Hospital.

First Septenniad, 1833—40	.	.	.	.	.	4,666
Second „ 1840—47	.	.	.	.	.	6,608
Third „ 1847—54	.	.	.	.	.	11,224
Total	.	.	.	.	.	<u>22,498</u>

The following Table will show the number of women confined in each month, during the twenty-one years :

TABLE I.

*Showing the Number of Women confined in each month, during  
Twenty-one Years, extending from 1833 to 1854.*

Months.	1st Septd.	2d Septd.	3d Septd.	Total.	Ratio.
October . . .	353	601	909	1863	8·24
November . . .	366	565	924	1855	8·20
December . . .	415	604	946	1965	8·83
January . . .	391	571	983	1945	8·58
February . . .	400	623	1012	2035	9·00
March . . .	412	576	1047	2035	8·79
April . . .	377	526	884	1787	7·97
May . . .	440	479	881	1800	8·18
June . . .	362	483	877	1722	7·64
July . . .	384	523	935	1842	8·12
August . . .	367	491	896	1754	7·79
September . . .	399	566	930	1895	8·45
Total . . .	4666	6608	11224	22498	100·00

By comparing the totals in this table it will be seen, that in *February* and *March* the maximum of deliveries have taken place, and in *June* the minimum. The months, in this respect, are as follows :

February—March.	October.	April.
December.	November.	August.
January.	July.	June.
September.	May.	—

Table II distinguishes the number of the confinement, whether first or second, &c., in 22,498 women, extending from 1833 to 1854.

TABLE II.

*Distinguishing the Number of the Confinement in 22,498 cases, extending from 1833 to 1854.*

No. of Confinement.	1st Septd.	2d Septd.	3d Septd.	Total.	Ratio.
First . . .	760	1022	1723	3505	15·68
Second . . .	743	1105	1845	3693	16·34
Third . . .	645	966	1622	3233	14·25
Fourth . . .	581	791	1442	2814	12·41
Fifth . . .	501	756	1185	2442	10·91
Sixth . . .	447	571	946	1964	8·85
Seventh . . .	335	455	795	1585	7·12
Eighth . . .	252	346	568	1166	5·20
Ninth . . .	154	232	422	808	3·52
Tenth . . .	110	149	300	559	2·40
Eleventh . . .	63	96	177	336	1·44
Twelfth . . .	38	55	81	174	0·78
Thirteenth . . .	15	33	35	83	0·36
Fourteenth . . .	9	12	32	53	0·19
Fifteenth . . .	5	8	21	34	0·13
Sixteenth . . .	6	6	4	16	0·08
Seventeenth . . .	—	2	6	8	0·03
Eighteenth . . .	—	1	3	4	0·01
Nineteenth . . .	—	—	2	2	0·00
Twentieth . . .	1	—	4	5	0·01
Twenty-first . . .	—	1	—	1	0·00
Twenty-second . . .	1	—	—	1	0·00
Twenty-third . . .	—	1	1	2	0·00
Twenty-fourth . . .	—	—	—	—	—
Total . . .	4666	6608	11224	22498	100·00

Table III distinguishes the *sex* in 21,553 children *born alive*; and Table IV distinguishes the *sex* in 1128 *still-born* children.

TABLE III.

*Distinguishing the Sex in 21,553 children born alive.*

Sex.	1st Septd.	2d Septd.	3d Septd.	Total.	Ratio.
Males . . .	2318	3290	5670	11278	52·32
Females . . .	2114	3015	5146	10275	47·68
Total . . .	4432	6305	10816	21553	100·00

TABLE IV.

*Distinguishing the Sex in 1128 children still-born.*

Sex.	1st Septd.	2d Septd.	3d Septd.	Total.	Ratio.
Males . . .	157	213	334	704	61·74
Females . . .	106	140	178	424	38·26
Total . . .	263	353	512	1128	100·00

From an examination of these tables it will be seen, that the proportion of males to females *born alive* is as 52·32 to 47·68, and the proportion of males to females *still-born* is as 61·74 to 38·26.

By comparing the two tables, it will also be evident that there is an excess of nearly  $9\frac{1}{2}$  per cent. in the *males still-born* as contrasted with the *males born alive*; and a corresponding deficiency of nearly  $9\frac{1}{2}$  per cent. in the *females still-born*, as compared with the *females born alive*.

It also appears that the ratio of children *still-born* to the children *born alive*, is 5·2 per cent.

Table V shows the nature of the labour in the 1128 still-births.

TABLE V.

*Showing the varieties of Labours in 1128 cases of still-born children.*

Labours.	1st Septd.	2d Septd.	3d Septd.	Total.	Ratio.
Vertex Presentation . .	103	186	243	532	46.35
Face . . . . .	6	4	1	11	1.17
Premature Labour . .	40	19	84	143	12.98
Perforation after induction of . . . . .	—	2	—	2	0.19
Breech Presentation . .	30	29	35	94	8.55
Foot „ . . . . .	16	25	44	85	7.24
Knee „ . . . . .	—	1	1	2	0.19
Shoulder „ . . . . .	5	1	6	12	1.12
Arm „ . . . . .	3	4	14	21	1.65
„ with Funis . . . . .	2	3	1	6	0.58
Funis Presentation . .	4	7	19	30	2.40
Placenta . . . . .	7	5	9	21	1.65
„ with Perforation . .	1	—	—	1	0.16
Thorax Presentation . .	—	1	—	1	0.16
Hydrocephalus . . . .	1	—	—	1	0.16
Delivered by Vectis . .	1	1	—	2	0.19
„ Forceps . . . . .	5	6	5	16	1.41
„ Perforator . . . . .	24	32	19	75	7.29
„ Secale . . . . .	4	—	—	4	0.50
Rupture of Uterus . . .	3	—	—	3	0.30
Flooding Labour . . . .	2	1	8	11	1.17
Puerperal Convulsions .	—	5	1	6	0.58
Twins . . . . .	6	20	12	38	3.40
Triplets . . . . .	—	1	6	7	0.49
Monsters . . . . .	—	—	3	3	0.30
Cæsarian Section . . .	—	—	1	1	0.16
Total . . . . .	263	353	512	1128	100.00



TABLE VI.

*Showing the Varieties of Labour in 22,498 women, attended between October, 1833, and October, 1854.*

Cases.	1st Septd.	2d Septd.	3d Septd.	Total.	Ratio.
Natural Labour. . .	4290	6218	10553	21061	93·61
Vertex Presentation . .	4266 } 24 }	6188 } 30 }	10512 } 41 }	20966 } 95 }	
Face " " . . .					
Premature Labour . . .	49 } 6 }	55 } 26 }	32 } 94 }	96 } 169 }	183
" induced . . .					
" Perforation . . .	— } 1 }	1 }	— }	13 }	1 }
Protracted Labour . . .	62	76	64	202	0·89
by action of Ergot . .	16 } 9 }	3 } 23 }	11 } 29 }	30 } 61 }	
" Forceps . . .					
" Vectis . . .	12 }	18 }	— }	30 }	
" Perforator . . .	25 }	32 }	24 }	81 }	
Preternatural Labour . .	109	116	271	496	2·20
Breech Presentation . .	59 }	60 }	117 }	236 }	
Foot . . .	29 }	34 }	80 }	143 }	
Shoulder . . .	6 }	4 }	5 }	15 }	
Arm . . .	6 }	7 }	36 }	49 }	
" with Funis . . .	3 }	3 }	— }	6 }	
Hand with Head, &c. . .	— }	— }	16 }	16 }	
Funis Presentation . .	6 }	4 }	16 }	26 }	
Knee . . .	— }	3 }	1 }	4 }	
Thorax . . .	— }	1 }	— }	1 }	
Complex Labour . . .	94	113	158	365	1·62
Twins . . .	33 }	49 }	104 }	186 }	
Triplets . . .	— }	2 }	4 }	6 }	
Placental Presentation .	13 }	13 }	16 }	42 }	
" with Perforation . .	1 }	— }	— }	1 }	
Retained Placenta . .	37 }	30 }	12 }	79 }	
Puerperal Convulsions .	4 }	11 }	10 }	25 }	
" with Twins . . .	— }	2 }	— }	2 }	
Hydrocephalus . . .	— }	— }	1 }	1 }	
Epileptic Mania . . .	1 }	— }	— }	1 }	
Puerperal " . . .	1 }	— }	— }	1 }	
Albuminuria . . .	— }	— }	2 }	2 }	
Hysterical Epilepsy . .	— }	— }	1 }	1 }	
Typhus Fever . . .	1 }	— }	— }	1 }	
Precidentia Uteri . . .	— }	— }	1 }	1 }	
Prolapsed Funis . . .	— }	4 }	— }	4 }	
" Bladder . . .	— }	— }	1 }	1 }	
Labial Thrombus . . .	— }	— }	1 }	1 }	
Lacerated Vagina . . .	— }	1 }	— }	1 }	
Undilatable Os . . .	— }	1 }	— }	1 }	
Monsters . . .	— }	— }	2 }	2 }	
Rupture of Uterus . . .	3 }	— }	3 }	6 }	
Flooding Labour . . .	51	50	80	181	0·80
Impracticable Labour . .	1	—	—	1	
" with closed Os . . .	1	—	—	1	
Spurious Pregnancy . . .	1	—	—	1	
Hydatid Placenta . . .	1	—	—	1	
Blighted Ovum . . .	1	2	—	3	
Died undelivered . . .	—	1	—	1	0·004
Cæsarian Section . . .	—	—	2	2	0·008
Total . . .	4666	6608	11224	22498	100·00

From an analysis of the foregoing table, it appears that 21,061 labours were natural, being in the proportion of 93·61 per cent. Of this number, the *vertex* presented, in 20,966, in the proportion of 99½ per cent. The cases of face presentations amount only to 95, or 0·45 per cent., or 1 in about 222 cases.

Of the 20,966 cases of vertex presentation, in 532 the children were still-born, in the proportion of 1 in 39, or 2·53 per cent.

In 11 of the 95 face presentations, the children were still-born, or 1 in 8·7, or 11·5 per cent.

The following table will show the duration of labour in 46 face presentations; in 49 the duration unfortunately has not been mentioned (most of these being omitted in the third septenniad).

Duration : hours 2, 5, 7, 8, 9, 10, 12, 13, 14, 15, 18, 20, 21, 22, 29.

Number of Cases, 1, 1, 2, 5, 1, 9, 9, 1, 6, 3, 4, 1, 1, 1, 1.

#### PREMATURE LABOURS.

By referring to the table it will be seen that 183 labours are reported as premature: of this number 14 only were induced; of the 169 that spontaneously occurred, 149 were still-births, or 88·1 per cent. The ratio of premature still-births to the total number of still-births is 14·09 per cent.

The following table will show the method of operating, the length of time that elapsed from the period of induction to the commencement of pain, and the completion of labour, the presentation, and event to child.

No.	Method of Induction.	Hours before occurrence of pain.	Hours before completion of labour after operation.	Presentation.	Event to child.
1	Puncture & Secale	27½	50	Nates.	Dead.
2	Puncture.	12	20	Feet.	"
3	"	"	40	Nates.	"
4	"	132	138	Vertex.	Alive.
5	Separ. of memb.	11	27½	"	Dead.
6	Puncture.	2	7½	"	"
7	"	27	40	Breech.	"
8	"	12	29	Vertex.	"
9	"	42	78	"	Alive.
10	"	90	102	"	"
11	"	57	69	"	"
12	"	35	44	"	Dead.
13	"	27	47	"	"
14	"	36	50	"	"

From this table it will be seen that only 4 children were born alive out of 14, and these were vertex presentations, and in which the operation of puncturing the membranes was performed; and it is also remarkable, that in the successful cases, the length of time after the operation, and before the occurrence of pain or completion of labour, almost doubles that in the unsuccessful cases; in fact, the sum of the hours in the 4 successful cases, exceeds by 152 the sum of the hours in the 10 unsuccessful ones.

One case proved fatal to the mother from the supervention of peritonitis; and in another, the operation of perforation had to be performed, and in this case the placenta was found partially adherent.

#### PROTRACTED LABOURS.

In 202 women the labour was protracted, being in the proportion of 0·89 per cent.

The chief causes which led to the protraction of labour were, uterine inertia, rigidity of the soft parts, disproportion between the head of the child and pelvis of the mother, and firm and extensive ossification of the foetal head.

In 30 cases out of 22,498, delivery was assisted by the *secale cornutum*, making a proportion of about 1 in 750 deliveries.

In 91 cases, delivery was effected by the embryospastic instruments: viz., 61 by forceps, 30 by *vectis*, or about 1 in 247·2 deliveries.

In 81 cases the operation of craniotomy was had recourse to, being in the proportion of 1 in 277·7 deliveries.

By adding the deliveries by the *vectis* and forceps to those by the perforator, there will be a total of 172 instrumental cases in 22,498 deliveries, or in the proportion of 1 case in about 130·8 deliveries.

In 31 cases the long forceps were employed, in the remaining 30 delivery was effected by the short forceps; 16 children out of the 61 delivered by forceps were still-born, and but 2 out of the 30 delivered by the *vectis*. The causes of perforation in the 81 cases in which this operation was had recourse to, were various degrees of pelvic deformity, ossification of the foetal head, adhesions and cicatrices in the vagina, disease

of the external genital organs, hydrocephalus, rupture of vessel in vulva with subsequent laceration of mucous membrane, puerperal convulsions. In 2 cases only did sloughing of the vagina follow ; but it was unattended in both cases with any fistulous communication with the rectum or bladder.

#### PRETERNATURAL LABOURS.

The cases of preternatural labour amount to 496, or 2·20 per cent. The number of *still-born children* amounts to 251, or 50·6 per cent. The *nates* presentations were 236, and of these 94 were still-born. The proportion of nates presentations *per cent.* as compared with the whole number of cases, natural or otherwise, attended during the 21 years, is 1·05. The *footling* cases were 143 in number, the still-births 85. The presentations of the *upper extremity* were 86, the still-births 39. The *funis presentations*, including prolapsus of the cord, amounted to 30 ; in 27 the children were still-born.

#### COMPLEX LABOURS.

The number of cases of complex labour amounts to 365, or 1·62 per cent. The number of still-births amounts to 84, about 1 in 4·3, or about 23 per cent. Of these 365 cases, 6 were triplet labours, 7 of the children still-born. For presentations, &c., see following tables :

The following tables will show the proportion of the sexes in *triplet labours*, and also the nature of the presentations :

No. of Cases.	All Males.	All Females.	2 Females and 1 Male.	2 Males and 1 Female.
6	1	1	2	2

#### PRESENTATIONS.

Vertex in all . . . . .	3	Two Vertex and 1 Foot . . . . .	1
One Vertex and 2 Breech . . . . .	1	Two Vertex and 1 Hand and Arm . . . . .	1

#### TWIN LABOURS.

These were 186 in number, or about 1 in 120·9 of the whole number of women delivered, being 0·82 per cent. The number of twins still-born amounts to 38.



The following table will show the proportion of the sexes in twin labours, and the nature of the presentations :

No. of Cases.	Both Males.	Both Females.	One of each Sex.
186	56	54	76

#### PRESENTATIONS.

Vertex in both . . . . .	84	Vertex and Hand . . . . .	3
Vertex and Nates . . . . .	33	Foot in both . . . . .	2
Vertex and Foot . . . . .	44	Shoulder and Foot . . . . .	2
Vertex and Shoulder . . . . .	4	Nates in both . . . . .	3
Vertex and both Knees . . . . .	1	Nates and Foot . . . . .	3
Vertex and Arm . . . . .	6	Vertex, Breech and Foot . . . . .	1

In the case of the twins presenting with the vertex and shoulder, the child presenting with the vertex was anencephalous.

In 43 cases "*presentation of the placenta*" has occurred. In 18 cases the placenta *entirely* covered the os uteri, while in 25 it *partially* presented. *Turning* was performed in all except three; 22 of the children were still-born. Seven of the cases terminated fatally to the mothers, principally from exhaustion; one, however, from pericarditis, and one from peritonitis.

#### RETAINED PLACENTA.

These cases, amounting to 93, include those where the placenta was retained from *uterine inertia*, and irregular uterine contraction, and those where there existed *morbid adhesion*. Forty-seven cases of the latter are recorded, and in the majority of these the placenta was adherent to the anterior and upper part of the uterus, as ascertained by the introduction of the hand to effect its removal. In the former class of cases the uterus was stimulated to contract by hand pressure, sometimes by the introduction of the hand, and occasionally the *secale cornutum* was exhibited.

#### PUERPERAL CONVULSIONS.

Twenty-seven cases of puerperal convulsions are reported, 4 occurring in the first, 13 in the second, and 10 in the

third septenniad; being in the proportion of 1 in about 833 cases.

The following table will show the number of confinement, age, method of delivery, sex of and event to child, event to mother, condition of urine, &c., in the various cases.

No.	No. of Confinement.	Age.	How Delivered.	Sex of Child.	Event to Child.	Event to Mother.	Condition of Urine.	Married or Unmarried.
1	1st.	21	Natural powers.	Not stated.	Living.	Recovered.	Not stated.	Unmarried.
2	5th.	36	"	Female.	" (7 mo.)	"	"	Married.
3	1st.	Not given.	"	Not stated.	Living.	"	"	"
4	9th.	"	"	"	"	"	"	"
5	1st.	20	Version.	Female.	Still.	"	Albuminous.	"
6	2d.	25	Natural powers.	Twins, (F.)	1 dead, 1 alive.	"	"	"
7	1st.	22	Perforator.	Male.	Dead.	"	"	"
8	9th.	32	Version.	"	"	Died.	"	"
9	1st.	18	Forceps.	Female.	Living.	"	"	"
10	2d.	22	Natural powers.	Male.	"	Recovered.	"	"
11	1st.	18	"	"	"	"	"	Unmarried.
12	2d.	25	Forceps.	Female.	Dead.	"	"	Married.
13	1st.	Not given.	Forceps & Version.	Twins, (M. & F.)	Living.	"	"	"
14	2d.	30	Natural powers.	Female.	"	"	"	"
15	4th.	28	"	Male.	"	"	"	"
16	2d.	25	"	"	"	Died.	"	"
17	1st.	31	"	Female.	Dead.	Recovered.	"	"
18	"	Not given.	"	"	Living.	"	"	"
19	2d.	27	"	"	"	"	"	"
20	1st.	19	Perforator.	Not stated.	Dead.	Died.	"	Unmarried.
21	"	24	Natural powers.	Male.	Living.	"	"	Married.
22	3d.	43	"	Female.	"	Recovered.	"	"
23	1st.	18	Forceps.	"	Dead.	Died.	"	"
24	11th.	50	"	"	Living.	"	"	"
25	1st.	28	"	"	"	"	"	"
26	No distinct report			—	—	Recovered.	—	—
27	No report.			—	—	—	—	—

It will thus be seen, that of the 26 cases reported, 8 proved fatal to mothers; 8 only of the children were still-born, 19 alive (there being 2 cases of twins), of the 25 fully reported cases; 6 of the children were delivered by forceps, 3 by version,

and 2 by the perforator; the remaining 15 were expelled by natural powers. Of the forceps and version cases, both operations were resorted to in one case of twins; 13 of the cases were first labours (3 being illegitimate), 6 were second labours. In one of these, the same woman is mentioned, as a primipara, in the table (No. 5), as having had convulsions in her previous labour; and by looking further down the table, to No. 15 (a fourth confinement), she appears again; and it is also stated in the report, that after her third confinement, she had a slight convulsive fit, accompanying a severe post-partum hemorrhage—the child in this case was a living female, and the urine albuminous. In another second confinement the labour was premature, and in two others the women had had convulsions in their previous labours; one was a third confinement, where the patient had never previously suffered from convulsions; one a fourth, in a patient who had had convulsive fits in each previous labour (the same woman mentioned above, in the second confinement); one a fifth, no statement as to previous convulsions, nor in the *two cases* of the ninth confinement; and one of the eleventh, in which the woman had been “*subject to fits*” for years.

It will be observed, on looking at the table, that the “*condition of the urine*” in the first 4 cases (first septenniad) is not stated, as the presence of albumen in the urine had not then been recognised in association with puerperal convulsions; but, in the last 21 cases, it will be seen, that, with one exception, the urine was albuminous, and in this case the convulsions were only a symptom of arachnitis (as discovered post mortem), and not the usual form of puerperal eclampsia. An analysis of the blood was made in only two cases (as reported), and in both of them a small quantity of the elements of urine was found.

#### HYDROCEPHALUS.

Three cases of hydrocephalus are reported during the twenty-one years, being in the proportion of about 1 in 7482 cases. All the patients recovered. One, however, suffered from peritonitis afterwards, but eventually got well. The other two recovered without a bad symptom, with the exception of some

difficulty in passing water for a day or two, requiring the use of the catheter. In all perforation was had recourse to.

#### RUPTURE OF THE UTERUS.

Seven cases of this distressing accident have occurred during the twenty-one years, together with 3 cases of supposed rupture. Of the 7 cases, all proved fatal; and of the 3 supposed cases, only one recovered; therefore, 9 out of 10 cases ended fatally. Six of the 7 presented by the head, the seventh by the elbow. In this last case, version was performed, and afterwards perforation. One woman was delivered by the forceps, and one patient died undelivered before any assistance could be procured. In 4 cases perforation was resorted to; in one after the operation of version, and in the remaining three the forceps had been first unsuccessfully applied. Of the cases examined necroscopically, the situation of the rent in the uterus was universally at its posterior part, mostly extending to the right side and posterior part of the cervix. In two cases it implicated the vagina. In all the cases, blood was found effused into the abdominal cavity, or between the layers of the broad ligament. In only one did any particular disproportion between the head and pelvis seem to exist. Of the three cases of supposed rupture, two presented by the head; in one of these, after an unsuccessful application of the forceps, turning was had recourse to, and the woman recovered. In the other head presentation, perforation was performed first, then turning; this was a fatal case. The third case presented by the feet, and was terminated by the natural powers. This woman also died. All the children were still-born, and one of them was putrid.

#### FLOODING LABOUR.

The number of cases amount to 181, or 0·84 per cent. Under this division are included 53 cases of *accidental* hemorrhage before the birth of the child, 60 cases of hemorrhage after the birth of the child but before the expulsion of the placenta, and 68 cases occurring after the expulsion of the placenta. In the 53 accidental cases, 3 children only are noticed as still-



born (some are not reported during the last septenniad), and rupture of the membranes in most cases was successfully practised. In the cases occurring before the expulsion of the placenta, manual assistance, aided by the exhibition of ergot, with compression, were successfully had recourse to.

# FATAL CASES.

The number of fatal cases to mothers, that have transpired during the twenty-one years, is 160, or in the proportion of about 1 in 140 cases.

The following table will show the *causes of death* in the various cases, with their proportionate numbers :

Puerperal Fever, Metro-phlebitis, } 96	Phthisis . . . . . 1
Peritonitis, &c. . . . . }	Arthritis . . . . . 1
Flooding . . . . . 21	Muco-enteritis . . . . . 1
Rupture of the Uterus . . . . . 9	Jaundice . . . . . 1
Convulsions . . . . . 8	Smallpox . . . . . 1
Pneumonia, Bronchitis, and Pleurisy 7	Mania . . . . . 1
Phlebitis and Phlegmasia Dolens . 4	Puerperal Epilepsy . . . . . 1
Cholera . . . . . 2	Labial Thrombus . . . . . 1
Impracticable Labour . . . . . 2	Malignant Disease of Uterus . . 1
Pericarditis . . . . . 1	Cæsarian Section . . . . . 1
	<hr/> 160

The great preponderance of cases under the head of puerperal fever, metro-phlebitis, peritonitis, &c., indicates the main source of danger to puerperal women, and one against the spread of which the greatest caution is preserved. This number embraces some epidemic attacks, and the difference in the terms by which it is designated is accounted for by the length of time over which the report ranges. The causes of death in the remaining cases do not require comment, as they are not in excessive proportion to the number of deliveries; and it is not our design in this statistical summary to add any observations of our own.

## SELECT CASES.

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BY H. M. HUGHES, M.D.

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### 1. CASES OF BLACK URINE FROM THE ADMINISTRATION OF CREOSOTE, &c.

GEORGE P—, æt. 14, by occupation a baker, and residing at Croydon, was admitted into the hospital under my care, August 15th, 1855. He was, in appearance, a thin and delicate lad, but was reported to have had good health up to the summer of 1854, when he had an attack of fever, and was confined for three months, but then got well enough to return to his work. His father died five years ago from erysipelas of the head. His mother is a delicate strumous-looking woman; but all her other children are healthy. About four months since, the patient first experienced severe pain across the abdomen, which was generally increased about an hour after taking food, and was sometimes accompanied with vomiting. He got gradually weaker, and became emaciated, and was, in consequence, admitted into Croydon Infirmary; but being no better after three months, he came to the hospital.

Upon admission, he presented the aspect of a weak and strumous lad. The light hair, the blue eyes, the delicate skin, the ill-nourished body, and the distended abdomen, all indicated a delicate constitution. The tongue was moist and clean, the appetite bad, the skin natural, and the bowels regular. He complained of pain and tenderness on pressure in the right iliac region, over a space the size of a saucer, which was resisting and crusty to the feel, and dull upon percussion, while the other parts of the abdomen were soft and resonant. He was ordered—Hydrarg. c. Cretâ; Pulv. Ipecac.

c., āā, gr. ij, nocte maneque; Ol. Morrhuæ, ʒij, bis die; with beef tea and arrow-root for diet.

In the course of a few days the oil was discontinued, in consequence of its causing vomiting. A blister, and afterwards a large poultice, was applied to the abdomen, by which means the pain was greatly diminished. But he still continued to emaciate, complained of pain above the pubes when his bowels were relieved and when the bladder was emptied, supposed to indicate peritonitic adhesions. The pills were continued for several weeks. The bowels were occasionally relieved by enemata, and his power was supported by milk and beef tea; and about a month after his admission the hardness and local tenderness in the right iliac fossa had in a great measure disappeared; but there was developed between it and the umbilicus a more superficial, more defined, and more doughy enlargement, which was believed to be some soft inorganizable deposit, the result of peritonitis, if not actually pus. To this was assiduously applied, every morning, the compound tincture of iodine, with the view of obtaining its absorption, and at the same time he was ordered Syr. Ferri Iodidi, ʒss, ex Inf. Quassia, ter die. The pills were to be taken at night only, and he was allowed half a pint of porter and four ounces of wine. About October 12th, after a variety of changes in his diet, to please his somewhat fickle temper and fastidious appetite, he suddenly became much worse; the left leg became œdematous, the emaciation rapidly increased, the eyes became sunken, and the expression anxious. Upon the 23d the tumour became more diffused, and he was attacked with sickness, vomiting everything that was taken into the stomach; for this sickness bicarbonate of potass and hydrocyanic acid were prescribed, and his port wine was changed for an increased quantity of sherry, but without any beneficial effect. He was, consequently, ordered a drop of creosote, in the form of a pill, every six hours. This had the result of effectually staying the sickness; but after taking it for three days the patient himself directed my attention to his urine, which was indeed peculiar, and such as I had never seen before. It was about ten ounces in quantity, and of a dirty or brownish black colour; clear; unaltered by heat, nitric acid, or by liquor potassæ, even when boiled with it. It was

without sediment, and remained unchanged in appearance after being kept in an open vessel for several days. It presented no traces of blood globules, or of any other solid substance, under the microscope. The poor lad died exhausted the next day.

*Inspection, forty-four hours after death.*—A catheter was first introduced into the bladder, and a few ounces of black urine drawn off similar to that passed during life. External appearances: *Rigor mortis* slight; emaciation very great; œdema of the left leg and thigh; some discoloration from decomposition of the walls of the abdomen. Chest: The apices of both lungs were sprinkled with miliary tubercles in groups, and some recent pneumonic deposit was irregularly distributed through the bases of the lungs. The heart was healthy. Abdomen: The cavity of the peritoneum was obliterated by the perfect adhesion of the whole of the contained viscera, the bond of union being infiltrated with strumous matter, which formed a layer over them in every part, and was in some places softened down so as to form small abscesses. This was particularly the case at the site of the external tumour, where there existed a larger amount of softened scrofulous matter than at any other part. Here, also, were one or two holes in the intestine, but whether they existed during life or were torn after death could not be determined. The liver was closely adherent to the diaphragm, and the spleen to the stomach, and each was covered with a layer of strumous matter. The structure of the former was fatty, that of the latter normal. The mucous membrane was puckered and softened at the cardiac extremity of the organ, where it adhered to the spleen, and the lymphatic glands were also full of strumous matter. The duodenum and jejunum were internally healthy, but the ilium was full of tubercular ulcers in all stages of formation. The large intestines were healthy, with the exception of a large, irregularly shaped ulcer in the sigmoid flexure of the colon. The mesenteric glands were full of scrofulous matter. The common iliac, the external iliac, and the commencement of the femoral vein of the left side, were filled by a clot of fibrin adherent to the coats of the vessel, and soft and whitish in the centre, which satisfactorily accounted for the œdema of the left leg and thigh.



The preceding case, though presenting many interesting features in connection with the affection of the peritoneum, would not have been considered worthy of any separate record excepting from the very remarkable state of the urine, on account of which alone it is here introduced. In the first instance I was disposed to believe the case to be novel, but in this was greatly mistaken, as my friend and colleague, Dr. Rees, who happened to be in the ward at the time while it was under examination, referred me to the great work of Dr. Pereira, on '*Materia Medica*,' wherein some cases by the late Dr. M'Cleod are quoted from the '*Medical Gazette*,'<sup>1</sup> in which cases black urine followed the administration of creosote soon after its first introduction as a therapeutic agent. In these cases, so much as  $\text{m}^{\text{xv}}$  of creosote was administered three times a day in one case, and in the other case the doses of  $\text{m}^{\text{ij}}$  were rapidly increased to  $\text{m}^{\text{xv}}$  three times a day. The medicine was then omitted, but in consequence of the return of the symptoms was resumed in a fortnight, in doses of  $\text{m}^{\text{v}}$ , increased in the course of eight days to  $\text{m}^{\text{xij}}$ , three times a day. The urine now became black, and it is to be presumed that it had not been of that colour before, or that it had not been observed to be so. Dr. M'Cleod describes the fluid as "something like natural urine to which a small quantity of Indian ink had been added;" and continues, "when this change in the urine was first observed, I carried some of it to Dr. Prout, to whom the fact was new, but who informs me that he has been unable to ascertain on what it depends."

Through the kindness of Dr. Wilks I have been referred to two earlier instances of black urine, recorded by the late Dr. Marcet, of Guy's Hospital, in the twelfth volume of the '*Medico-Chirurgical Transactions*,' p. 37, and occurring in the year 1814, a period long anterior to the employment of creosote as a remedial agent. It is described as "quite black and opaque, though without any sediment or turbidness, and on the surface, when examined in a strong light, a dark purplish hue was discernible, giving to the liquid the appearance of a strong solution of the extract of liquorice. This urine had been discharged by a healthy male child, of the age of seventeen months." It seems to have occurred almost im-

<sup>1</sup> *Vide* '*Medical Gazette*,' vol. ii, for 1834-35, p. 599.

mediately after birth, to have "been passed perfectly clear, and gradually to have assumed the black colour. The phenomenon, moreover, admitted of occasional variation in degree, and even sometimes entirely disappeared." "The child, nevertheless, always enjoyed a good state of health." It was, after the age of seventeen months, entirely lost sight of. Dr. Marcet also referred to an instance of black urine occurring in a patient of his at the City Dispensary in the year 1802, in the person of a young female who laboured under a very singular and anomalous disorder, lasting between two and three months, after which it was entirely removed.

Dr. Wilks has also preserved and kindly supplied me with notes of the following cases. The peculiar urine in the former of the two I did not see, as it was unfortunately thrown away by the nurse, and did not reappear.

*Case of Fever accompanied with Blue Urine.*

William E—, æt. 18, was admitted into the hospital under my care, July 5th, 1853. He stated that he was a sailor, and had been ill during the whole of his voyage from Rotterdam, which had extended over two weeks, and that he had been on shore four days before his admission, when he had simple fever, of considerable intensity, with drowsiness, much tremor, regular bowels, and without rash. On the 15th he began to get better, and on this day, while taking wine and ammonia, the chamber-vessel was observed to be full of a bright blue urine, passed during the night. The fluid was covered with a pellicle of the same bright blue colour, which remained adhering to the sides of the vessel when the urine was poured out. It was, unfortunately, thrown away by the nurse before any analysis was made, and no more was subsequently passed.

*Case of Strumous Peritonitis, in which a Black Deposit occurred upon boiling the Urine with the addition of Nitric Acid.*

Emma B—, æt. 11, admitted under my care May 31st, 1854. She had been ill for three or four years with gastric fever, diarrhœa, and other complaints referrible to the abdominal organs. On admission, the abdomen was tumid, hot, and tender. The urine was examined for albumen, but none was

discovered; but, upon boiling it with nitric acid, a brownish, or purplish-black deposit occurred, which rendered it both opaque and turbid. The same result occurred on several occasions, but ceased altogether before she was removed from the hospital, on the 28th of June. The child lived only three or four weeks after returning home. Upon inspection after death, most extensive strumous disease of the peritoneum and intestines was found, the folds being inextricably bound together by adhesions, and the mucous membrane being extensively ulcerated. There was also commencing disorganization of the lungs. The kidneys were quite healthy.

Differently from the case of the sailor suffering from fever just recorded, who passed blue urine, in this, as in the case first related, abundant opportunities were enjoyed by both microscopists and chemists for examining the black material; but they all informed me, that they were unable to determine either its nature or its cause, excepting that it appeared to be a peculiar form of colouring matter, probably carbon.

My friend Dr. Hermann Weber wrote to me on March 24th, 1856, as follows: "You were sometime ago so kind as to show me a dark brown urine passed under the influence of creosote. I have obtained just now some very similar from patients who are rubbing tar over the whole body. Of three cases that I have lately treated in this way, this change in the urine took place in two only, while in the third the colour of the urine remained always light. As yet, I must add, I have not been able to find the cause of this peculiar colour, although I have tried various means to do so."

Dr. H. Weber has since informed me, that he has procured creosote from this dark urine by distillation; and that, in one case, the urine first passed after the inunction with *tar* (not tar ointment) was black.

We have then herein noticed nine cases of coloured urine, or—excluding the young woman, a patient of Dr. Marcet, with the "singular and anomalous symptoms," who, from not being constantly under supervision, it is quite possible to conceive might have intentionally produced it by some admixture; and excluding also the case of bright blue urine in the sailor recovering from fever, in whom the fluid was not chemically or



microscopically examined, but in which the colouring matter was, in all probability, dependent upon the presence of indigo, as pointed out by Dr. Hassall—we have seven cases noticed in which the urine was of brownish or purplish-black colour. In three of these cases creosote had been administered internally; of which in two it had been given in doses of from five to twelve, and even fifteen drops, three times daily; but in the other in a dose of only one drop four times a day; and when it is recollected, how very often this remedy has been administered in the hospital in doses equally large or larger, this effect must be considered as remarkable. In two cases tar had been applied externally for psoriasis, and creosote was obtained from the urine by evaporation; while in the case of the infant related by Dr. Marcet, it is highly improbable that it or any similar substance had been taken; and in the case of the little girl mentioned by myself, it assuredly had not been prescribed. In six of the seven cases the urine was passed black and clear, and remained unaffected, or but slightly affected, by chemical reagents, including heat and nitric acid; whereas, in the seventh case, it was passed of a natural appearance, and only threw down a dense black precipitate upon the addition of nitric acid and the application of heat. Dr. Odling, who examined this urine, states, “that the black precipitate, on subsequent exposure, became an indigo-blue deposit.” This fact was certainly not observed by myself, or by the pupils attending the case; but it appears to me to be a very interesting fact in connection with the circumstance mentioned by Dr. Hassall in his paper “On the frequent presence of Indigo in the Human Urine,”<sup>1</sup> in which he says, “that for the formation of this (blue) indigo, it is in general necessary that the urine should be exposed to the air for some days in an open vessel, when oxygen is absorbed, and the blue indigo developed;” and also with the statement made by Dr. Odling, in his note to me, that “creosote and indigo belong to two closely allied chemical families.” It may be necessary, however, to remark, that, while in all the cases of blue urine related by Dr. Hassall in his paper, the blue colour appeared only after the exposure of the fluid for some days to the action of the air, from which certain decompositions and fresh chemical combinations pro-

<sup>1</sup> *Vide* ‘Philosophical Transactions, vol. cxliv, part ii.



bably occurred, in all the cases of black urine noticed in this paper, excepting one, it was black when it was passed; and that it is at least highly probable, that the blue urine herein mentioned was passed of that colour, as the fluid passed during the night was found blue in the morning.

P.S.—Since writing the above, the following passage has been pointed out to me, in the paper of Dr. Elliotson on the “Medicinal Properties of Creosote,” in the nineteenth volume of the ‘Medico-Chirurgical Transactions,’ p. 237: “The woman began the remedy in November. Her urine became black in February, and remained so a short time. I was not informed of this circumstance till after it had ceased, and did not see the urine; and I understand that other patients taking the remedy in the hospital have occasionally experienced the same effect for a short time. Some private patients have described the colour of the urine as green.” Upon what circumstance it depends that the administration of creosote in some cases should cause the urine to be black, and in a vast majority of other cases it should induce no such effect, remains to be explained, and I regret to state that I have no explanation to offer.

## 2. CASE OF FATAL INTUSSUSCEPTIO ASSOCIATED WITH LUMBRICI.

*From the Report of Mr. A. D. Brooks.*

Daniel D—, æt. 14, was admitted into the hospital under my care, February 27th, 1856. He resided near the Tower, and assisted his father, who is a tailor. His previous health had been very good. Seven weeks ago he was exposed to great cold, and on the following morning he was seized with severe pain in the abdomen, which lasted for several hours, and then ceased, but returned the next day. This pain had continued to recur at uncertain periods up to his admission. He had been attacked twice in one day, and the longest time that he had been free was from the 21st to the 25th of February, when he took some castor oil. From that time he had suffered from tenesmus, loss of appetite, and vomiting after meals. He

described the pain as a twisting and tearing of his intestines, principally about the umbilicus; and during the paroxysm he could himself feel lumps in the abdomen, which disappeared when it had passed. During the paroxysm he lay with his legs drawn up, and pressed his hands upon the abdomen with relief. The duration of the fits was very variable; they now generally recurred upon alternate days, were frequently dispelled by the passage of flatus, and when they were absent he felt quite well. His appetite was capricious; his bowels were usually relieved twice in a day, and the motions were natural. On admission he was considerably emaciated, with remarkably prominent cheek-bones, narrow forehead, and a dreary careworn expression of the eyes; his tongue was white, furred, and moist; he was suffering from severe colicky pains; the abdomen was rigid, tender in some parts when pressed, and free from tenderness in others, lumpy and uneven from irregular distension of the intestines during the paroxysms, but soft, flaccid, and free from any tenderness when they were absent. The pulse was weak and compressible; the skin was natural. He passed a "lumbricus teres," together with some mucus, soon after his admission. Ordered—Fotus Papaver. abdomini; Hydrargyr. Chlorid., gr. v.; Opii, gr. ss. M. ft. Pil. statim sumend. Haustus Sennæ, hor. iv post pil. sumend., et in hor. vj repetend. Milk diet.

28th.—Had no sleep during the night, and was this day in great pain, though his bowels had been efficiently relieved. Ordered—Enema Terebinth. statim; Hydrar. Chlorid., gr. j; Opii, gr.  $\frac{1}{4}$ , 6tis horis; Haustus Sennæ tepidus; nocte.

29th.—Had passed a quiet night, and slept well. He was now quite free from pain, and the abdomen flat and flaccid generally, though a hardness could be felt in the left hypochondrium. His bowels had been freely opened, and he had passed another worm; he had had no return of vomiting, and his appetite was "ravenous." Rep. Pil., et cràs mane Haust. Sennæ c.

March 2d.—His bowels were sufficiently acted on. He had been again troubled with the same colicky pains, but not nearly so severely as formerly, nor can the rolls of intestines be so distinctly traced as formerly. Ordered to omit all medicine and to have middle diet.

4th.—Exhibited a good deal of depression, and the bowels were sluggish. Ordered—Julep. Ammoniae (of the Hospital), Decoct. Aloes c., āā ʒss ter die.

Nothing worthy of notice occurred in the reports till on the—

10th.—The pains had returned, and he appeared to suffer considerably, although an aperient previously administered had operated freely, and had brought away another lumbricus. Ordered—Pil. Galbani c., gr. v, ter die sumend., together with the Mist. Magnes. c. Magnesiae Sulphate (of the Hospital); Enema tepidum statim.

11th.—Had not found any relief, and the pains had been more severe than at any time since his admission. Ordered—Hydrar. Chlorid., gr. j; Opii, gr.  $\frac{1}{4}$ , ter die; Enema Assafoetid.

14th.—Yesterday and this morning he had been attacked with diarrhœa; the motions being, according to his own report, of a “slimy” or mucous character. Ordered—Pulv. Cretæ c., gr. xv, ex Julep. Ammoniae (of the Hospital Pharmacopœia).

15th.—Had suffered greatly from pain, evinced by the pinched and haggard expression of countenance; he had no rest during the night. The tongue was now coated with a white fur; the pulse small, quick, and compressible; the abdomen distended, rolls of intestine being clearly visible on its surface, and appreciable by the touch, and tender upon pressure. He now appeared to be suffering from the ordinary symptoms of dysentery; the evacuations, voided with considerable tenesmus, consisting of bloody mucus without any fæcal matter. He had lost his appetite altogether, and had vomited a quantity of bilious fluid. Ordered—Hirudines, iij, ano applicand.; Enema Amyli c. Liq. Opii sedativ. ʒxv, statim injiciend.; Haustus Olei et Rhei (of the Hospital Pharmacopœia), ʒss, post hor. iij; Fetus Papaveris abdomini; Mist. Acaciæ, ʒj, Liq. Opii sedativ. ex Julepo Menthæ, ter die. Milk and suet for diet.

18th.—Little alteration had taken place in the symptoms; the suffering had been excessive, and he was greatly exhausted by the extreme pain. He had now incessant vomiting of green bilious fluid, and severe pain in the abdomen, which was distended, but always wavy, from contracted rolls of intestine, one being especially obvious in the left iliac region; the tongue remained foul. Suspicions were entertained that



some improper articles of food, to say the least, were brought in by his friends, and they were consequently forbidden to enter the ward. The ejected matter was analysed, but no trace of any mineral irritant could be discovered, nor could any suspicious article of food upon diligent search be found in his locker. Ordered—*Olei Filicis maris*, ʒj, primo mane, et *Olei Ricini*, ʒss, post hor. iv; *Liq. Opii sedativ.* ℥viij, *Æth. Chlorici*, ℥viij, ex *Mist. Camph.*, 6tis horis sumend.

19th.—The whole of this morning the vomiting continued incessantly, and, indeed, up to 3 p.m., at which time he ejected another lumbricus by the mouth, to the great disgust and commiseration of the nurse and patients of the ward, after which the sickness was alleviated. In the evening, however, the abdominal pain was most severe, and he passed bloody mucus by the bowels, with much tenesmus, but without any fæcal matter. Ordered—*Opii*, gr. ss, 4tis horis. Rice pudding, and wine, ʒiv.

20th.—The injection passed freely into the bowel without causing any pain, and was retained a considerable time, but was at length returned without fæcal matter. The sickness was now trifling, but the abdominal pain most severe, and though the abdomen was tender upon pressure in some parts, it appeared not to be the tenderness of peritonitis; it was uneven, lumpy, and hard at some parts, and soft and flat at others; but at all times, I believe, there was more or less hardness and tenderness on pressure at the scrobiculus cordis and in the left hypochondrium. Ordered—*Rep. Pil. Opii*; *Fellis bovini*, ʒj; *Decoct. Avenæ*, ʒxv. *M. ft. enema statim injiciend.*

22d.—The injection passed freely as before, and returned with some fæculent matter. He was in much less pain, indeed tolerably comfortable, but complained of great exhaustion, and looked greatly depressed and emaciated from his suffering. *Rep. Enema Fellis bovini.* *Rep. Pil.*; wine, ʒvj.

23d.—Having left him tolerably comfortable yesterday, we were greatly surprised and annoyed to find him this morning greatly depressed, suffering from tense abdomen, tender upon pressure, extreme pain, with a pinched face, anxious countenance, clammy skin, and a barely perceptible pulse, in fact, with all the symptoms of perforated intestine; symptoms which



appeared to have supervened suddenly, soon after the visit of his friends yesterday afternoon, a circumstance which, after our former suspicions, could not fail to attract notice. He died a few hours after the visit.

Before giving an account of the post-mortem examination, it may be observed that a variety of opinions were given as to the nature of this very interesting and perplexing case. By one party it was thought that the whole symptoms, up to the last fatal collapse, might possibly be induced by the irritation of worms, of the existence of which we had proof; by another that some malignant disease was the original source of the symptoms; by a third that some irritant was administered to the lad by his friends; and by a fourth that the disease was intussusception.

*Inspection, twenty hours after death.*—Rigor mortis present; body wasted. The head was not examined. Chest: There were old, firm pleuritic adhesions of the left lower lobe; in other respects the organs of the chest were healthy. Abdomen: It was rather full, but not inordinately distended. General peritonitis existed, lymph covering the intestines, and effused yellow fluid existing in the dependent portions of the cavity. The inflammation had been most acute in the inferior part of the cavity, there being but little lymph upon the surface of the liver or of the diaphragm. A small portion only of the intestines was observable, and this was red, softened, and presented the ordinary characters of inflammation. The inflammation appeared to be due to a recent perforation which had taken place in the sigmoid flexure of the colon. When the omentum was cut through, it was seen that almost the whole of the intestinal canal was disarranged and misplaced. No cæcum or ascending colon could be discovered, and upon tracing down the ileum, it was found to end abruptly in the transverse colon, a little to the right of the mesian line. Upon removing the effused lymph and separating the adhesions, an intussusception was discovered, by which the whole ascending colon, the cæcum, and an unascertained portion of the ileum, had passed into the transverse and descending colon and rectum. Another peculiarity was observed. Upon cutting through the great omentum, a sac was discovered between the stomach and colon, about the size of the stomach itself. To

this sac an entrance was found below the transverse colon, and in it were contained more than half of the upper portion of the small intestines, almost empty, and quite free from inflammation, while all or almost all the other organs of the abdomen were affected with peritonitis. In consequence of twisting of the intestines and various adhesions, it was difficult to determine how this peritoneal sac between the stomach and the colon was formed. But so far as could be ascertained under the circumstances, it appeared that the ascending colon had never been fixed to the posterior wall of the abdomen, but had, together with the cæcum and termination of the ilium, been entirely surrounded with peritoneum, and consequently free like the small intestines; and also that some deficiency had existed in the meso-colon, through which the greater portion of the small intestines had passed, beneath the transverse colon, into the sac of the omentum. The serous membrane, however, which directly enveloped them, appeared to be formed of the ascending portion of the meso-colon itself. The transverse colon, the descending portion, and the rectum were greatly distended, in consequence of the invagination of the intestines, which could be felt to extend almost to the anus itself. Upon removal of the parts, the lower part of the ilium was seen entering the colon rather to the right of the mesian line; the latter bowel, the descending colon, and sigmoid flexure being thrown into numerous transverse folds, so as very materially to diminish their length, and to render it probable that the ascending colon was really the commencement of the including portion of intussusception, and also, after the parts were separated from their connections, to remove the included portion some inches from the anus. Upon laying open the extremities of the sheath of bowel, the invaginated intestine was slightly bent upon itself, from the traction of the mesentery. The whole was of a blackish-green colour, which denoted commencing gangrene. At the inferior extremity considerable ulceration of the mucous membrane had taken place. The opening of the included canal was not in the centre, but, owing to traction, was directed to one side, and of a linear form, but readily admitting the point of the finger. The passage, though small, was patent throughout, and contained a small quantity of faecal matter, as did the intestine below it. The lower extremity of

the included bowel lay, while extended after removal, in the sigmoid flexure; and, in corresponding parts, the two portions of bowel, included and including, were extensively ulcerated. It was in the latter that the perforation had occurred which had given rise to the fatal peritonitis. At the upper extremity, on the contrary, recent adhesions, barely separable, had taken place between the contained and the containing intestines, which, together with the approaching gangrene, and a line of ulceration at the upper extremity of the former, appeared to indicate the commencement of a process for its entire separation from the body—a process which has been previously observed and successfully effected in some other cases of a similar nature. The mucous membrane of the lower part of the small intestines, situated in the general cavity of the peritoneum, was of dark purple colour from congestion, and was ulcerated in two or three places. Several lumbrici were contained in them. The other organs were healthy. (See Plate II.)

In a case presenting so many anomalies and difficulties, it would be futile to speculate as to the original cause of the symptoms, or as to the time and the succession of events; as it is probable that no two pathologists would accord in opinion upon these questions. When the internal hernia occurred; whether it preceded or followed the intussusception, or whether even it was a congenital condition, it is quite impossible to determine. Viewing the case, however, as one of simple intussusception, certainly of many days' duration, it may be observed as somewhat remarkable that no pure blood was passed—the sole tendency to such an escape existing in a little bloody mucus, analogous to an ordinary dysenteric stool; that obstruction of the bowels was not permanent; and that the injections were always passed with ease, and administered without pain. These circumstances, not common in intussusception, may, I think, be attributed to the fact of the canal of the included bowel being not only perfectly permeable, but also to its lower extremity being so near to the anus as to be dilated by the injection, or possibly even to allow of the introduction of the clyster-pipe.



### 3. CASE OF EMPHYSEMA OF THE ABDOMINAL PARIETES FROM PERFORATION OF THE RECTUM.

Joseph B—, æt. 50, a widower, and a lighterman, was admitted into Philip Ward, under my care, May 14th, 1856. For the first few weeks after his admission, he complained mainly of vomiting, constipation, pain after taking food, accompanied with emaciation and great depression of spirits. These symptoms had continued, with greater or less severity, for four months, and had been but little relieved by the treatment previously adopted. He was supposed to labour under some organic disease of a malignant character, in all probability of the stomach itself, though no evidence of any tumour could be discovered by manual examination. It is unnecessary to record the succession of symptoms usually present in such cases, or the variety of remedies administered, which, in this as in other instances, were more or less effectual in relieving symptoms, but nothing more.

Nothing worthy of particular notice occurs in the report of Mr. W. Smith until June 10th, when an injection was ordered to be given. The patient had had several before, administered by the nurse of the ward, without any pain or inconvenience whatever. He would not, however, on this occasion, allow her to introduce the gum elastic tube, believing that he could do it very well himself. The consequence of this was, that, in the act of passing it, he experienced excruciating pain, and withdrew the tube without injecting the fluid. He soon complained of pain in the abdomen, of a spasmodic character, for which a mustard poultice was applied. The next morning, about fifteen hours after the accident, upon manually examining the abdomen, we were surprised to find air under the skin. It was not generally diffused, but existed in small patches, varying from the size of a sixpence to half a crown; crepitated sensibly under the finger, and very loudly under the stethoscope. It was not till some days afterwards that we were made acquainted with the accident, and were therefore in the interim unable to account for the emphysema which continued, but did not materially increase, except that it extended to the



parietes of the lower parts of the chest. Several injections were subsequently administered by the nurse, without pain. When made acquainted with the accident, and suspecting it to be the cause of the emphysema, Mr. Callaway was requested to examine the rectum, and reported that a portion of sloughing membrane was protruding from the anus, quite black, and of a tape form, and, moreover, that the rectum was so tender and the attempt to examine it caused so much pain, that the patient refused to submit to it.

The next day (June 20th), the report states that he was much purged, and so much exhausted as to be unable to get up to defecate. The discharges were excessively offensive and of a gangrenous odour, and, when capable of being preserved and seen, looked, with the exception of a few olive-coloured scybala, like a portion of the enemata administered, combined with unhealthy purulent secretion. Occasionally he passed a small fluid evacuation unmixed with any injected fluid. It then looked like dirty, fetid pus, and by the addition of liquor potassæ was changed into characteristic, clear, viscid mucus. Ordered to repeat the opiate enema, and to have a chloride of lime poultice.

24th.—There had been a great improvement since the last report: the sickness had almost ceased, the diarrhœa had stopped, and the purulent discharge had diminished; the anxiety of countenance, formerly present, had disappeared; his appetite had improved, he slept well at night, the power of the pulse had increased, and the emphysema was no longer to be felt.

July 2d.—The bowels had become again confined, he passed more pus per anum, sickness had returned, and he had lost his appetite. Ordered — *Haust. Olei et Rhei, 3vj, st., et Enema commune.*

From this date the sickness continued unabated; he gradually sank, and died at 6 a.m. of the 6th.

*Inspection, thirty-two hours after death.*—The head was not examined. With the exception of the lungs being emphysematous, nothing worthy of record was observed in the chest. Abdomen: The stomach was greatly dilated and its coats much thickened. The pylorus was much contracted, barely admitting the point of the finger, and upon division

was found to be half an inch thick from carcinomatous deposit, as well as from hypertrophied tissue, and upon the duodenal side was an ulcer the size of a sixpence, with elevated edges. The lymphatic glands in the vicinity were much enlarged by cancerous deposit. The intestines were quite healthy, with the exception of the rectum, the lower part of which was greatly distended, and almost the whole of its inner surface was occupied by a large ulcer, which was four inches in length and three inches in breadth, leaving only a narrow strip of mucous membrane, about an inch wide, at the upper part. The edges were raised and vascular, and the surface of the ulcer was ragged and composed of bands of muscular fibres with hollow spaces between them. Beneath one thick band of muscular fibre was a round opening which passed into the cellular tissue in the ischio-rectal fossa. It was, probably, through this that the pipe passed so as to give rise to the emphysema. The kidneys were small and granular; the other organs were healthy.

The sequence of events in this case appears to be so clear as not to admit of any doubt. The patient, in the act of passing an enema-tube, experiences great, indeed, excruciating pain; fifteen hours afterwards he has emphysema of the integuments of the abdomen; a few days subsequently he has offensive purulent discharges, and a portion of sloughing membrane passed per anum; and after death a large ulcer of the mucous membrane of the rectum is discovered, together with a perforation of the muscular coat. It appeared probable that a large abscess had formed between the mucous and muscular coats; that the former had sloughed away, leaving the latter bare, while the cellular membrane behind it had been comparatively little affected. I believe that cases of perforation of the rectum from the unskilful introduction of clyster-pipes are not very rare, and that most museums contain specimens of the accident, but I do not recollect myself to have met with an instance in which it was productive of emphysema.

p.s.—While these pages are at press, I have a man under my care in the hospital, in whom the urine is rendered of a black colour when boiled with nitric acid. A further analysis shows this condition to be due to iodine, and it need scarcely

be said that the patient is taking this remedy internally. To a similar cause, no doubt, must be referred the dark urine in the case of Emma B—, mentioned at p. 56, who was also under the same treatment.

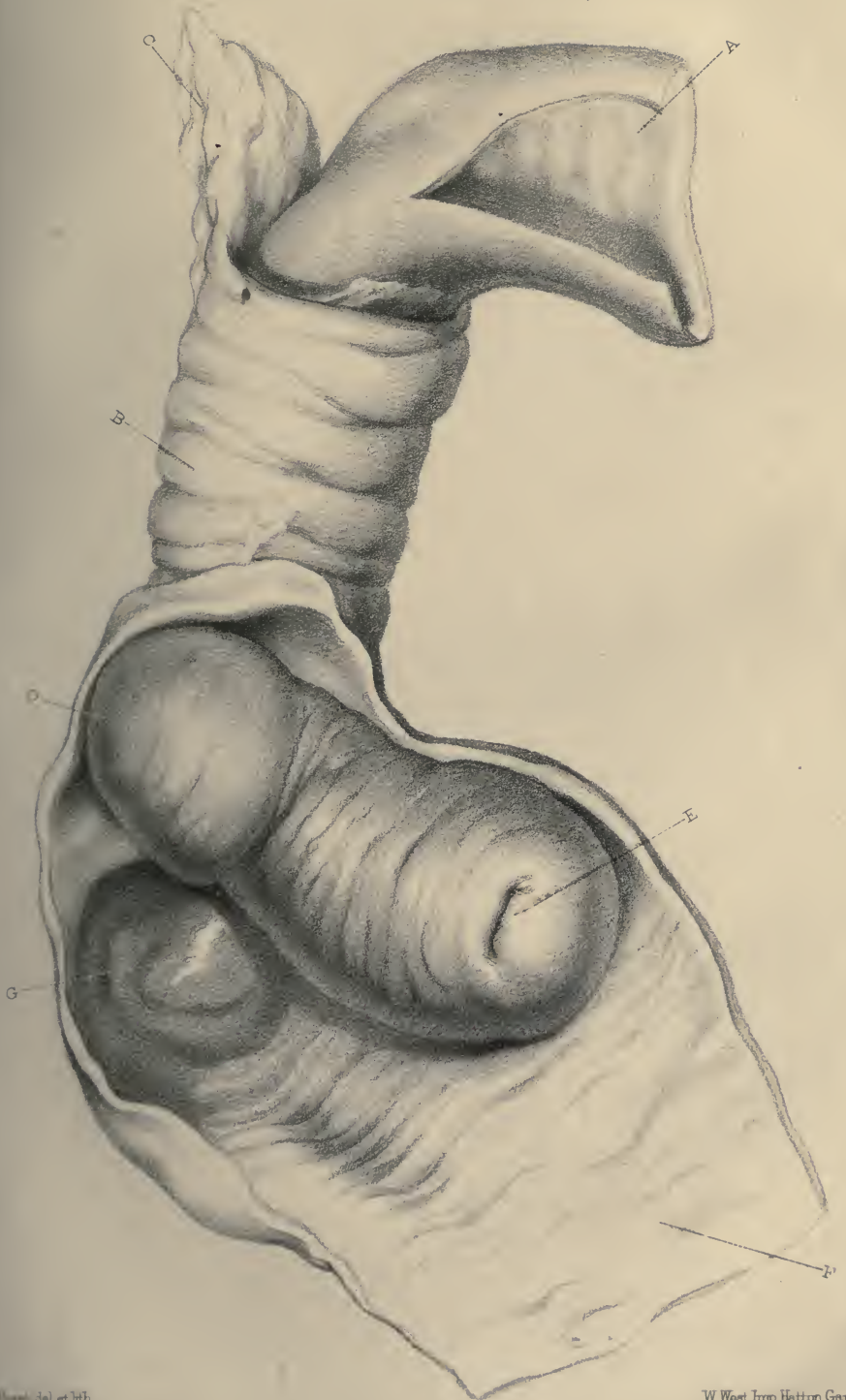
Time does not allow me to put this explanation to the proof, by testing the urine in other cases of patients who are taking iodine, but I have little doubt that it is the correct one,—that is, the medicine probably produces this change under certain conditions of disease.

## PLATE II.

Representing Dr. Hughes's case of Intussusceptio.

- A. The ileum ; the end of which, with the cæcum, has passed through the corrugated colon, seen at B.
- C. A portion of meso-colon.
- D. The bulbous extremity of the invaginated intestine brought to view by laying open the colon.
- E. The natural opening at the extremity of the intussusceptio.
- F. The rectum cut off near the anus, and turned aside to show E and G.
- G. The ulcerated opening in the sigmoid flexure, which gave rise to the fatal peritonitis.







ON HERNIA,  
WITH  
AN ANALYSIS OF 126 FATAL CASES.

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BY THOMAS BRYANT.

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WHAT form of hernia is most common? What form most frequently requires operation? And which is most fatal? are questions which constantly arise in the minds of all surgical students.

To aid in the solution of these questions, the following pages have been written, and towards the execution of the last, 126 fatal cases have been collected from the records of Guy's Hospital, the reports of which are good and authentic. In 88 of these, the particulars of the post-mortem examinations are preserved; and from these alone have been drawn up the tables and inferences as to the causes which have produced a fatal termination.

A. As regards the first question, *What form of hernia is most common?* the statistics of the London Truss Society, ending with the year 1855, most satisfactorily determine; for, out of 84,478 cases of inguinal and femoral hernia, 75,054 belonged to the former, and 9424 to the latter division: or, as it is more clearly stated in per centages, out of every hundred cases, 88·8 per cent. were inguinal, and 11·1 femoral; inguinal hernia being most frequent by 77·7 per cent. (*Vide Appendix.*)

B. Towards the solution of the query, *What form of hernia most frequently requires operation?* 281 cases have been collected; of these 105 were inguinal, and 176 femoral: or, in

every hundred cases, 37·4 were of the former kind, and 62·6 of the latter. Femoral hernia, therefore, required operation 25·2 per cent. more frequently than inguinal.

c. With respect to the third question, *What form is most fatal?* two tables have been framed—one of successful cases, and one of fatal.

In 169 *cures* after operation, inguinal bore the proportion to femoral as 36·1 per cent. to 63·8, the difference being 27·7 per cent. *in favour* of femoral hernia.

In 117 *deaths*, inguinal bore the proportion to femoral as 44·4 per cent. to 55·5, only 11·1 per cent. being the difference *against* femoral hernia. But as the difference in recoveries was 27·7 per cent., it is clear that inguinal hernia is more fatal than femoral by 16·6 per cent.

d. We now come to the analysis of the 126 fatal cases of hernia; and, bearing in mind the fact that tables are only valuable in so far as, by their analysis, they enable us to establish, or bring out, certain conclusions, it has been thought undesirable to occupy valuable space by their publication as a whole. The materials, however, upon which the various conclusions are founded, have been compiled into many short tables, enabling others thus to examine the grounds upon which these conclusions are based. These tables having been framed with great care, and each conclusion carefully weighed before written, it is hoped that the truth of each will be acknowledged, and the omission of the table, as a whole, not considered unwise.

TABLE I.

*Short Analysis of the 126 Fatal Cases.*

		Males.	Females.	Sac.		Side.	
				Opened.	Not.	Right.	Left.
126	65 Femoral . . .	13	52	32	19	34	14
	52 Inguinal . . .	52	—	30	4	16	17
	8 Umbilical . . .	6	2	3	1	—	—
	1 Obturator . . .	—	1	—	1	1	—



E. *The ages at which the different forms of hernia first make their appearance.*—From the following table, it will be observed, omitting the congenital form, that, in inguinal hernia, from 20 to 40 years of age is the period when the rupture most frequently commences; but in femoral at these ages it is comparatively rare, as, in the majority of cases, it appears between 50 and 70 years of age.

TABLE II.

	Inguinal.	Femoral.		Total.
	Males.	Females	Males.	
Congenital . . . . .	7	—	—	7
Age of 10 years and under . . . . .	2	—	—	2
" 20 " " . . . . .	4	1	—	5
" 30 " " . . . . .	8	4	—	12
" 40 " " . . . . .	5	8	1	14
" 50 " " . . . . .	1	7	1	9
" 60 " " . . . . .	2	11	6	19
" 70 " " . . . . .	1	9	2	12
" 80 " " . . . . .	—	3	—	3

F. *Duration of the hernia before strangulation.*—In 28 cases, it was recent, and strangulated on its first descent. In 98 cases, it had existed years. Recent cases bearing the proportion to old as 22 per cent. to 77. Of the 28 recent cases, 21 were femoral and 7 inguinal; and of the 21 femoral, 17 were about 60 years of age. From this it is evident that femoral hernia is more frequently strangulated upon its first descent than inguinal.

Seven of the 98 cases were congenital. Fourteen had existed *many* years, the exact number not being stated.

Of the 76 remaining—

TABLE III.

	Inguinal.	Femoral.		
	Males.	Females.	Males.	
10	2	7	1	had existed 5 years or less.
11	2	5	4	
17	8	6	3	
5	3	1	1	
3	3	—	—	
				" 10 "
				" 20 "
				" 30 "
				" 40 "

The average duration of femoral previous to strangulation being 11 years ; of inguinal, 20 years.<sup>1</sup>

G. *Ages when the hernia became strangulated.*—The succeeding table by its evidence well supports the conclusions drawn from Tables II and III. In the former, it was shown that inguinal hernia most frequently made its first appearance between the ages of 20 and 40 ; and femoral, between the ages of 50 and 70. In the latter, that the average duration of inguinal hernia prior to its strangulation was 20 years ; of femoral, 11. From this it would be expected, that the favorite ages for inguinal would be between 40 and 60 ; and for femoral, between 60 and 80. And this the table fairly proves, remembering at the same time the fact, that femoral hernia is most liable to strangulation on its first descent ; and that of the 28 recent cases, 21 were femoral, and that the subjects of it were about 60 years of age.

TABLE IV.

	Inguinal.	Femoral.		Total.
		Females.	Males,	
Age of 20 and below . . . . .	1	1	—	2
„ 30 „ . . . . .	14	3	—	17
„ 40 „ . . . . .	9	7	—	16
„ 50 „ . . . . .	11	12	—	23
„ 60 „ . . . . .	10	13	5	28
„ 70 „ . . . . .	7	11	4	22
„ 80 „ . . . . .	—	5	3	8
„ 90 „ . . . . .	—	—	1	1

The average age of persons suffering from strangulated inguinal hernia being 43 ; from femoral, 55.

<sup>1</sup> An interesting paper by Mr. King, upon this subject, may be here referred to, published in the third volume of the first series of the 'Guy's Hospital Reports.'

II. *The period of strangulation* is the next point in the series of events, and this also is compressed into a table.

TABLE V.

20 cases were strangulated 24 hours, or less.			
26	"	"	48 "
15	"	"	about 3 days.
12	"	"	" 4 "
6	"	"	" 5 "
9	"	"	above 5 "

The minimum period of strangulation being 8 hours; the maximum, 14 days. The average period of strangulation for femoral hernia being 76 hours and a half; for inguinal, 50 hours and a half.

1. *Duration of life after operation.*—From the following table, it will be seen that more than half the cases died within 48 hours; the second day proving the most fatal; and that four fifths died within the first week. The most rapid death after operation being five hours, the operation having been performed merely as a *dernier ressort*.

TABLE VI.

Cases.	Inguinal.	Femoral.		
	Males.	Females.	Males.	
21	13	8	—	died within 24 hours.
35	15	17	3	" 48 "
10	3	7	—	" 3 days.
11	4	4	3	" 4 "
5	3	1	1	" 5 "
3	2	—	1	" 6 "
2	1	—	1	" 7 "
12	5	5	2	" 14 "
6	—	5	1	" 21 "
4	1	3	—	" 28 "

K. *Causes of death.*—The annexed table has been framed to show briefly the causes of death.

TABLE VII.

5	cases	died	without operation.
14	„	„	after the application of the taxis.
27	„	„	never rallied after the operation.
12	„	„	sank from diarrhœa, or collapse after a copious stool.
16	„	„	with artificial anus.
4	„	„	with internal strangulation.
2	„	„	after reduction of hernia <i>en masse</i> .
12	„	„	with faecal extravasation.
26	„	„	with general peritonitis and inflamed bowel.
1	„	„	with internal hemorrhage.
2	„	„	from sudden collapse.
5	„	„	from bronchitis, pneumonia, or erysipelas.

L. *Cases which died without operation.*—Of these 5, 2 were femoral, 2 inguinal, and 1 umbilical.

CASE I.—With the umbilical there was no history, except that the hernia had not been strangulated for more than twenty-four hours before death, but had existed for ten years. Gangrenous and firmly constricted omentum, and small intestine, formed the contents of the sac. No lesion, or appearance of disease, was found in any other portion of the body. The shock to the system had evidently been the cause of death.

CASE II.—Inguinal hernia, in a man, æt. 25; was similar to the last as to the cause of death, was without a history, and had been undiscovered. The testicle, descended only to the external ring, obscured a small congenital bubonocoele, consisting of omentum and ileum; these were congested and inflamed; the bowel above the strangulated portion was remarkably distended, and measured ten inches in circumference. The other viscera were healthy.

CASE III.—A scrotal hernia, in a man, æt. 34; who died on the fifth day after its strangulation. Acute peritonitis of the upper bowel, without effusion, existed, and a gangrenous sac containing faeces with gangrenous ileum and omentum. No internal extravasation was present, but, on applying traction



to the strangulated bowel, fæces flowed from the lacerating line of stricture.

CASE IV.—Femoral, in a woman, æt. 52 ; who died exhausted on the eleventh day, without the slightest evacuation from the bowels. The sac burst in separating the inflamed and nearly perforated skin, exposing an opening in the lower ileum, firmly adherent by its margin to the mouth of the sac ; flakes of lymph were scattered over the peritoneum, but no fæcal extravasation.

CASE V.—Also femoral, in a man, æt. 70 ; who sank exhausted from sloughing of the groin and copious intestinal discharges from the wound and anus, on the eighteenth day after strangulation. On the eleventh the sac had evidently given way, and on the twelfth an incision into the tumour was sanctioned by the patient ; fæces had passed *per rectum* for several days. The sac contained omentum, and patulous ileum, a portion only of its circumference having evidently been strangulated. The peritoneum was congested, but uninflamed.

M. *Fatal cases following reduction by the taxis.*—Of the 14 cases, 8 were inguinal and 6 femoral, 2 of the latter being males.

In 5 of these, 1 only being inguinal, the bowel was ruptured. In 2 (inguinal) the hernia was reduced *en masse*. In 2 (inguinal) strangulation still existed in the sac. In 1 (inguinal) internal strangulation existed. One died from diarrhœa on the second day, and one with peritonitis ; both were femoral, and in both gangrenous intestine was found.

In 2 cases, the peritoneal coat alone was ruptured. One, femoral, was admitted moribund, and lived only a few hours after its reduction ; the second, inguinal, survived two days, sinking with peritonitis.

N. *Ruptured bowel.*—In 2 of the 5 cases, extravasation followed, and speedy death. In 2 a flake of lymph prevented extravasation. In 1 (inguinal) the intestine was glued to the peritoneum, the man being much intoxicated ; after its reduction he walked to his bed, speedily collapsed, and died.

(Chloroform was not used.) In 3 of these cases death followed in a few hours; in 2 from extravasation, and in 1 from collapse after a copious motion. In 1 case, although the patient rallied after the taxis, she gradually sank on the fourteenth day; no peritonitis was present, the only abnormal condition being a portion of ileum, which had been ruptured, and its edges inverted and glued together.

*o. Cases which refused to rally after the operation.*—Amongst these 27 cases, 6 were inguinal, 19 femoral, and 2 umbilical.

12 died within the first 24 hours.

12    "    "    second    "

3    "    "    third    "

The average period of strangulation in these cases was fifty-two hours in the inguinal, and seventy-six in the femoral.

*p. Cases of death from diarrhœa, or collapse after a copious stool.*—Twelve cases are tabulated under this heading. Six were inguinal, and 6 femoral. Eight sank within twenty-four hours after its first appearance, and 6 of these rapidly after the *first* discharge. Four only survived, three, four, and five days respectively. In 4 the diarrhœa appeared within twenty-four hours after the operation; in 2 within two days; in 1 within three; 1 within four; and in 4 upon the fifth day. From this it is clear that collapse and death after a copious motion is by no means a rare occurrence.

*q. Artificial anus.*—Included in the 17 cases of artificial anus, were 13 femoral, 3 inguinal, and 1 umbilical.

In 8 of these the intestine was slit open at the time of operation: in 1 the gangrenous bowel was left in the sac; in 6 the bowel was returned into the abdomen, and subsequently sloughed.

In 2 (inguinal) no operation had been performed.

Three of the 8 cases where the bowel was slit open by the surgeon, lived two days; 2 three days; 3 twelve, fifteen, and twenty-five days, respectively; the last case went on well till the day of his death, when, on making some slight effort, he fainted and died.

In the case of gangrenous bowel which was left in the sac the intestine gave way on the third day, profuse faecal discharge followed, succeeded by collapse and death.

In 1 of the 6 cases where the gangrenous bowel had been returned into the abdomen, the sac was not opened at the time of operation; the patient was an old woman, æt. 72, collapsed at the time of operation; on the ninth day, the sac and intestine sloughed, and, upon the nineteenth, she sank exhausted. In 4 others, the faecal discharge first appeared upon the fourth, sixth, ninth, and tenth days, respectively; in all, death followed upon the ninth or tenth day, from exhaustion. In 1, acute peritonitis occurred after some weeks' favorable progress, from faecal extravasation.

From the above, we may fairly conclude that artificial anus much more frequently follows femoral than inguinal hernia. That the cause of death, in such cases, is generally exhaustion; and, that it occurs more rapidly when the intestine is slit open at the time of operation than when it naturally follows its return into the abdomen.

R. *Cases of internal strangulation.*—Of the 6 cases of hernia associated with internal strangulation, 4 were inguinal, 1 femoral, and 1 umbilical.

In 1, inguinal, the portion of ileum below the hernial protrusion was twisted upon itself: herniotomy had been performed. In 1, a band of membrane, passing from the mesentery to the omentum, was the cause of the strangulation: in this case the hernia had been reduced by the taxis. In 1 case of femoral, the cæcal appendage formed the constricting band. In the remaining 3 cases the hernia had been reduced *en masse*: in 2, inguinal, the tumour had existed for upwards of thirty years, in patients above 60, and 1 had been irreducible: one of these lived three days, the other eight, after its strangulation, and two and three after its reduction by the taxis; in both, peritonitis was the cause of death, associated in one with gangrenous bowel; in the other the gut was only incarcerated. The third case was umbilical, in a man aged 86. After four days' strangulation, herniotomy was performed, and the contents of the opened true sac reduced. On the third day after the operation death ensued, when recent peritonitis was found,



with a small omental sac, strangulating a piece of small intestine, the line of stricture only, being of an ash colour, and easily torn.

s. *Death from internal hemorrhage.*—This case occurred in a man, æt. 26, with inguinal entero-epiplocele; the omentum was incised, and intestine returned. On the second day the man died, when about a pint and a half of fluid blood was found in the peritoneal cavity; the epigastric artery was not wounded, the bleeding having evidently proceeded from the incised and reduced omentum.

t. *Cases of death from sudden collapse.*—Two cases are stated to have suddenly collapsed and died.

One, a man, æt. 73, with femoral hernia of twenty-two years' duration, was operated upon, and progressed favorably till the ninth day, when he suddenly collapsed, and died in a few hours. A low form of peritonitis was found on examination, and the sac inverted into the abdomen; to this was attached a dark knuckle of ileum, and at the point of junction the intestine was ash-coloured.

The second case, a man, æt. 39, after herniotomy for inguinal hernia, progressed well for ten days, and then suddenly sank. On necroscopic examination, a local peritonitis was found to exist at the mouth of the sac, glueing the reduced, congested, but elastic ileum, and inclosing a little puriform deposit; the mucous lining of the bowel was much congested, and covered with lymph. Viscera otherwise healthy.

u. *Contents of the sac.*—Of the 88 cases where the body was examined after death—

TABLE VIII.

In 57 the sac contained ileum.

2	„	„	„	with colon.
1	„	„	„	with cæcum.
6	„	„		jejunum.
22	„	„		small intestine, which portion not mentioned.
33 cases the omentum was included.				

In 4 of the 57 cases where ileum was the portion of bowel



strangulated it was the *upper* portion, in 3 the *middle*, and 21 a portion of the last two feet.

v. *Position of the contents, with respect to the neck of the sac.*

—In 9 cases they were adherent to the cavity of the sac ; in 40 cases they were adherent, or situated at the sac's mouth ; in 2 only they were stated to be distinct from the sac, and in these it may be questioned whether the manipulations in the examination were not the cause of their altered position.

w. *Condition of the hernia contents.*

TABLE IX.

In 31 cases the bowel was in various stages of congestion and inflammation, but *elastic*.

14	"	"	"	covered with lymph.
7	"	"	"	thickened.
15	"	"	"	gangrenous.
14	"	"	"	sulcated at the line of stricture.
17	"			artificial anus existed.
14	"			the bowel was perforated.
2	"			the peritoneal coat was alone ruptured.
6	"			faecal extravasation existed.

x. *Condition of the peritoneum.*

TABLE X.

In 10 cases it was declared to be healthy.

10	"	"		simply injected.
10	"	"		found greasy.
37	"			lymph was effused, more or less generally.
5	"	"	"	locally.
10	"			bloody serum was effused.
4	"			faecal extravasation had taken place.
2	"			fetid serum existed.

From the four preceding paragraphs, it is fair to conclude, that—

The ileum is by far the most frequent portion of the intestine strangulated, and that in the majority of cases it is part of the last two feet.

That the returned bowel, as a rule, remains at the neck of the sac, and that it is generally fixed there by adhesions.

That in about 69 per cent. of fatal cases, peritonitis exists sufficient to produce death, that is to say, lymph in some of its forms is generally effused.

y. *Gangrenous intestine, &c.*—Eleven of the 15 cases, tabulated in Table IX, were femoral, and 4 inguinal.

In 3 of the latter the hernia had been reduced by the taxis, and in 1 no operation had been performed. In *all* these cases the gangrenous bowel was situated at the mouth of the sac.

In Paragraph q it may be seen that there were 7 cases in which the bowel had been returned after operation, and in which artificial anus subsequently appeared. All of these cases were also femoral.

Of the 8 cases also of artificial anus made at the operation, 6 were femoral, 1 inguinal, and 1 umbilical. Omitting this latter, there are 29 cases of gangrenous bowel, 25 of which were femoral, and 4 inguinal; the cause of gangrene in these cases of inguinal having been evidently forcible taxis. From this it is clearly proved that femoral hernia is much more frequently associated with gangrenous bowel than inguinal.

z. *Bowel sulcated at the line of stricture.*—Under this head are 14 cases; 7 of these were femoral, and 7 inguinal. In 4 only, and these were inguinal, did ulceration occur at this line; in 3 general extravasation followed, and in 1 this was local.

zA. *Perforated bowel.*—Of the 15 cases, 10 were femoral, and 5 inguinal. In 4 cases the perforation occurred at the line of stricture; in 11 at about the *centre* of the knuckle. Of the 4 cases where it occurred at the line of stricture *all* were inguinal; in all ulceration was the cause; in 3 general extravasation followed, in 1 local. In 5 of the 11 cases where perforation took place about the centre of the knuckle, the taxis had been the cause of the rupture, and in 2 of these general extravasation followed. Four of the 5 cases were femoral, and 1 inguinal; in 3 (femoral) gangrene was the cause, and in 1 local extravasation followed. In 3 (femoral)

the perforation was produced by ulceration. In the remaining 8 cases extravasation was prevented by the exudation of lymph; in 4 a flake closed the aperture; in 2 the intestine was adherent to the peritoneum; in 1 case the bowel, perforated to the size of a pinhole, was left in the sac; and in 1 the edges of the rupture were inverted and glued together.

*zB. Fæcal extravasation.*—Three of the 7 cases in which fæcal extravasation occurred were femoral, and 4 inguinal; in 5 it was general, and 2 local. In 2 cases the taxis had been the cause; in 4 cases ulceration; and in 1 gangrene. The 4 cases in which ulceration had been the cause were all inguinal, and in all the upper line of stricture was the seat of the perforation.

*Conclusions.*—1. That *inguinal* hernia is more common than femoral by 77·7 per cent.

2. That *inguinal* hernia most frequently commences between 20 and 40 years of age, and *femoral* between 50 and 70.

3. That the average duration of inguinal hernia prior to its strangulation is 20 years, of femoral 11.

4. That inguinal hernia most frequently becomes strangulated before 50 years of age, and femoral after this time.

5. That the average age of persons with strangulated inguinal hernia is 43, of femoral 55.

6. That femoral hernia requires operation 25·2 per cent. more frequently than inguinal, success in its reduction by the taxis being less frequent.

7. That, after operation, inguinal hernia is more fatal than femoral by 16·6 per cent.

8. That 22 per cent. of cases requiring operation are recent, and are strangulated on the first descent.

9. That three fourths of these recent cases are *femoral*.

10. That the average period of strangulation in fatal cases of inguinal hernia is  $50\frac{1}{2}$  hours, of femoral  $76\frac{1}{2}$ .

11. That half the fatal cases of hernia die within 48 hours after the operation, and four fifths within the first week.

12. That three fourths of the cases which refuse to rally after operation are *femoral*.

13. That collapse and death after a copious motion is by no means a rare occurrence.

14. That artificial anus much more frequently follows *femoral* than inguinal hernia.

15. That the cause of death in cases of artificial anus is generally exhaustion; that death occurs more rapidly when the intestine is slit open at the time of operation, than when the opening in the bowel naturally follows its return into the abdomen.

16. That sudden collapse and death occasionally occur in cases of hernia which have progressed favorably for many days, without any *local* cause.

17. That in about 69 per cent. of fatal cases, peritonitis exists sufficient to produce death; that is to say, lymph in some of its forms is generally effused.

18. That in nine tenths of the cases of hernia, the *ileum* is the portion of bowel strangulated; and in three fourths of these it is part of the last two feet.

19. That as a rule, the strangulated bowel, when returned, rests or is fixed by adhesions at the mouth of the sac.

20. That in *all* cases of gangrenous bowel the affected portion will be found at, if not adherent to, the mouth of the sac.

21. That femoral hernia is much more frequently associated with gangrenous bowel than inguinal.

22. That ulceration at the line of stricture is more frequent in inguinal hernia; although the sulcated condition of the bowel is as common in femoral as in inguinal.

23. That fæcal extravasation, if not produced by ruptured bowel from the taxis, generally follows ulceration at the line of stricture, and is consequently generally found in inguinal hernia.

24. That ruptured bowel from the taxis is most frequent in femoral hernia.

25. That fæcal extravasation does not necessarily follow rupture of intestine by the taxis.

26. That fæcal extravasation does not occur so often as 50 per cent. in cases of rupture or perforation of the bowel.

From the above conclusions it is clear that —

*Inguinal hernia* is more common than femoral; commences earlier in life; is less liable to strangulation on its first descent,



and generally exists twenty years before it becomes so ; requires operation less frequently, but is more fatal ; is less frequently associated with gangrenous bowel, but more frequently with ulceration at the line of stricture.

*Femoral hernia* is less common than inguinal ; seldom appears before 50 years of age ; is more frequently strangulated on its first descent, but generally averages eleven years' existence ; more frequently requires operation, but is less fatal ; and is generally strangulated for a longer period before relieved. After operation, femoral hernia is more frequently followed by sinking than inguinal ; is more frequently succeeded by gangrenous bowel and artificial anus ; but is less frequently associated with ulceration at the line of stricture and fæcal extravasation, unless this occur from rupture by the taxis, to which it is *more* liable.

#### PRACTICAL DEDUCTIONS.

The preceding conclusions, brought out as they have been by a careful analysis of the materials collected—and each of which, it is believed, will be acquiesced in as facts by all who take the trouble carefully to peruse these pages—must necessarily yield (what all researches should invariably tend to) some practical points, either to guide us in the future practice of our profession, or to warn us from dangers, palpable or obscure.

1. The first point that may be dwelt upon, and which appears as clear as it is important, is the necessity of operating earlier in femoral than in inguinal hernia. The danger of delay in all cases, where strangulation is evident, is now so well appreciated by surgeons generally, that there is no necessity to stimulate such a feeling ; but, unfortunately, there is need to urge them to act upon the knowledge of it ; for the fact that so many cases are admitted into Guy's Hospital, in a dying and almost hopeless condition, proves too truly that such is not sufficiently understood, or at any rate is not practically carried out. But in femoral hernia delay is of peculiar importance, for three fourths of the cases which refuse to rally after operation are of this kind. This fact alone is sufficient

to indicate, that in femoral hernia, the ultimate result of all strangulation, namely, death of the part, occurs more rapidly than in inguinal. But when with this it is considered that this form is generally found in old women, who are, of course, less able to resist any such injury; that gangrenous bowel and artificial anus are much the most frequently associated with it, the truth (for such it must be called) that femoral hernia passes into a state of gangrene more rapidly than inguinal, becomes evident; and, as a result, the practical conclusion remains to be drawn, that in femoral hernia early relief is most imperative. And here the form in which it is to be applied must not be passed over; for recognising the conclusion that ruptured bowel from the taxis is most frequent in femoral hernia, together with the fact just mentioned, that gangrene is most frequently associated with it, the inference that the taxis should be employed sparingly and carefully cannot but be drawn. Our rule, then, in such cases, should be, to operate early; to let nothing like force be employed in the attempt at reduction, and not to let valuable time be lost by baths and other fancy remedies. To support also such an opinion, another argument may be employed, deduced from the conclusions numbered 1 and 6. It will be there seen that femoral hernia is less common than inguinal by 77·7 per cent., and yet requires operation 25 per cent. more frequently; leading us most fairly to infer that, when down, the taxis seldom succeeds in its reduction; and coupling this with the preceding remark, the practical conclusion just drawn becomes more apparent in its truth. The anatomical reasons for this, it might not, perhaps, be difficult to explain; but, as it is only intended to make remarks upon points which the materials collected seem to warrant, it will suffice to say that the smallness and unyielding nature of the crural ring, together with the sharpness of Gimbernat's ligament, seem sufficient for its explanation.

2. With these anatomical conditions are closely connected in interest another point, which certainly at first sight appears strange. In the twenty-second conclusion of the table, it is stated that ulceration at the line of stricture is more frequent in inguinal, although the sulcated condition of the bowel at the line of stricture is as common in femoral hernia. The

materials for drawing such an inference (Parag. z and zb) could warrant no other conclusion, and it can hardly be stated to be a chance that the cases collected should indicate such a result; for although the numbers of each species in which sulcated bowel was detected were equal, in no one case of femoral did ulceration exist; in several commencing gangrene coexisted, and in one rupture from the taxis, but not in the line of stricture. That such ulceration does occur it is not to be denied; but, as a rule, the tightness of the stricture seems so intense that death of the part is the general result; the more general and less rapid constriction of inguinal hernia, being, apparently, required to produce ulceration. That such an opinion is reasonable, analogy well bears out; for in surgery the application of a ligature to an artery, a nævus, or a polypus, if well and firmly applied, is first followed by sphacelus of the distal growth, and subsequently by ulceration, but if timidly applied, and without producing a total arrest of the circulation, ulceration will be the first if not the only consequence.

In the bowel, then, why should a different termination be expected? The general smallness of strangulated femoral hernia, and the anatomical peculiarity of the ring, acting and reacting upon one another, are rapidly followed by gangrene (unless relieved), and then ulceration. But in inguinal hernia the larger and more dilating neck, the greater mass of bowel which generally exists, affording, by its size, as it were, a cushion for the resistance of the former, is as a rule more frequently succeeded by ulceration.

3. That collapse and death after a copious motion is by no means a rare occurrence, is a conclusion perhaps recognised by all; but there is a point connected with it which may be considered worthy of a few remarks, and also of some practical importance.

The administration of purgatives after herniotomy is now, happily, obsolete; but the anxiety of the surgeon to witness a return of the action of the bowel, has not, perhaps, so subsided, as to prevent a frequent, and may be too frequent, use of the enema: that such is the case, occasionally, is not to be doubted, for the too frequent remarks in notes of reported



cases, such as "collapse and death after a copious stool following an enema," forbid any other conclusion. The subsequent treatment to herniotomy by opium is chiefly vaunted (and with perfect justice) for its value in preserving rest to the returned bowel, and thus allowing it time to regain its tone before being called upon to perform its natural duties. And here the question presents itself—Why urge it then at all? The lower bowel, on the first appearance of strangulation, is not unfrequently emptied of its contents, and seldom is it found after death at all distended with *fæces*; not so the upper portion. As a rule this will be seen full of liquid excretion, ready as it were to "flush" the lower intestine the moment the returned bowel has sufficiently recovered to permit its passage. Why, then, administer enemas? for although their action is not to be compared to the mischief produced by purgatives, still they do stimulate the intestinal canal, and unquestionably at times hasten, if not cause, death. That they are of value, and necessary in some cases, cannot be questioned: still as a rule, perhaps, they should not be administered, and certainly not merely to hasten the action of the bowel, unless decided symptoms are present which denote its over-distension.

4. In conclusions 19 and 20 it will be seen that, as a rule, the strangulated bowel, when returned, rests, or is fixed by adhesions, at the mouth of the sac; and that in all cases of gangrenous bowel, the affected portion will be found at, if not adherent to its mouth. If this latter is a rule, as it doubtless is, with only such rare exceptions as every rule possesses, the practice of stitching the edges of a bowel, when found in a gangrenous condition and opened, to say the least of it, is unnecessary. The object of such a practice, viz., to prevent the retraction of the divided portion from repassing into the abdominal cavity, and thus producing *fæcal extravasation* and its attendant consequences, being happily prevented. The inflammatory action which always attends strangulation of the bowel, and more especially at the sac's neck, carefully provides for this contingency, which it foresees; rendering not only *fæcal extravasation* from gangrene, in these cases, exceptional, if not unknown, but the practice of mechanically fixing the edges of the divided bowel unnecessary, and even wrong. For



the primary object for which such practice is generally adopted, having been shown to be needless, nature's processes having forestalled man's inventions, it remains for us only to reflect that in the second stage of artificial anus, that is, when the fears of extravasation, &c., have passed away, and the fact that the divided bowel and the sac's neck are firmly glued together is established, that this very retraction is the process by which any cure can be expected; for by it the ends of the divided bowel, with the retracting neck of the sac to which they are adherent, forms that temporary channel through which the intestinal fluids find their way to the lower bowel, and thus allow time for the other operations of nature to perfect a cure. Any interference, then, to such a process, cannot but be regarded as improper; and undoubtedly the ligaturing of the bowel's edges to the integuments must come under such a heading. For it is clear, if the necessary amount of retraction in the most favorable cases is but just sufficient to allow a cure, that the required portion of internal retraction must be almost entirely prevented, where the integuments and external coverings form the boundary, and with it the resistance. It is true that these assist well to form a firm and direct channel for the conduction of the fæces externally; but this very truth is an extra argument against the practice; for what hope can be entertained of a cure, when the bowel is, as it were, everted, and the traction has been exerted from without instead of from within. It may be said, however, that the practice forms an increased security, and that the chances of recovery, in artificial anus, being so slight, that it outweighs in the balance any objections against it. But to such reasoning it must be answered, that where security exists, extra security is not wanted; and that the fact that recovery from such cases of artificial anus does take place, should totally prevent the surgeon from pursuing a practice which excludes all hope of such a result, and at the best can only prolong a life which is generally a burden, and undoubtedly a misery to the patient and those around him.

5. With regard to the great question of opening or not opening the sac in hernia, the conclusions have a decided tendency towards the latter operation, although more, perhaps, by negative than positive evidence.

Not entering upon the mere question of per centage recoveries, as compared in the two operations—so many extra circumstances separately influencing the termination of a case, besides the question of opening or not opening the sac,—the present materials will only lead me to examine into the truth of the dangers by which the advocates of the former method believe the more lenient operation to be surrounded. And summed up into two heads, the fear of returning a ruptured or gangrenous intestine into the abdomen, or of a bowel strangulated by the sac's contents, include the principal, if not the only dangers. That such accidents may happen there is no doubt; but whether they are more liable to occur in such cases as may require an operation, than in cases where a line of practice is adopted which all agree upon as being the first means to be employed, namely, the taxis, is not so certain. To return a gangrenous bowel into the abdomen, either by the taxis, without dividing the stricture, or with doing so, is not easy. The accompanying inflammatory action not only always glueing the gangrenous intestine to the sac's mouth, but very frequently to the sac itself; thus rendering the application of the taxis in both its modifications perfectly useless. Here, then, the opening of the sac is called into requisition. But why, as a rule, do more than is required at an operation, because exceptional cases may occur, in which some fancied or real mischief may follow the simpler means? By the same argument, the taxis itself would be abolished, and herniotomy always be employed; for, undoubtedly, the former has produced more mischief, as a whole, than any operation, whether of opening or not opening the sac. But if this fancied mischief can be shown to be groundless, and if it can be shown to be probable that on the gangrenous bowel being returned, little or no evil is likely to result, the argument against such a practice falls to the ground, and, as a sequence, the milder operation is supported. Such a truth the materials of this paper seem to ratify; for in no one case of gangrenous bowel did extravasation occur, and in all, the intestine was glued to the sac's mouth. In many cases the bowel subsequently sloughed, but in all artificial anus followed; and in one instance, which made great impression at the time, the sac was *not* opened, the intestine was returned to its position at the sac's mouth,

subsequent sloughing followed, the sac easily gave way, and artificial anus was established; showing, at least, in one case, the leaving of the sac unopened was certainly not disadvantageous. In all the deaths related, not one case is numbered, where the opening or not opening the sac could have been a point of any importance. In the cases of gangrened bowel, it has been shown that no evil was the result of its return; but in conclusion 15 it is rendered probable, that the return of the gangrened bowel into the abdomen, and the leaving to nature the mode of establishing an artificial anus, was of a decidedly beneficial character. For it is certain, that in those cases where such a result followed the surgeon's interference, death followed *more* rapidly than where the process was left to nature. In the former cases, it may be said that such a result would be expected, as they were evidently of a worse character, or else such interference as opening the bowel would not have been deemed necessary. But such a conclusion is hardly just; for, as the danger of a strangulated hernia depends upon the obstruction to the intestine, and as that obstruction exists as surely from a gangrenous bowel within the abdomen as one within a hernial sac, and as relief in both first consists in the establishment of an external outlet till such an end ensues, the mechanical obstruction still exists, and both classes of cases are in an equal position. And as it has been shown that life is longer when nature's processes bring about this result, the advantages of returning a gangrenous bowel to the sac's mouth cannot be denied.

6. What, then, are the conditions of intestine where its return would not be advantageous? is a question which naturally follows; and with all due deference to the opinions of others, and with diffidence in making known my own, the force of evidence compels me to the conclusion, that in only one condition can such practice be deemed advisable, and that is where the bowel is decidedly ruptured. When gangrenous only, the neck of the sac has been shown to be its right position, in any other more favorable condition no one will dispute the practice of reducing it. But when ruptured, I would say leave it in its place, return any unaffected bowel if present, but leave the ruptured portion. And as the "let alone" practice



seems most favorable, even then, perhaps, it may be employed with benefit, and the bowel left for nature to open further. Here, then, research and analytic evidence come forward to support an opinion stated to the author by his late respected teacher, Mr. Aston Key, who startled him with the then strange opinion, "that in all conditions of the intestines the abdomen was their right place." That such a rule is right, excepting in case of rupture or perforation of the intestine, the force of the evidence compels me to admit; and although not expecting others altogether to agree, still, as the object in starting was fairly to express the results which the materials, together with much careful reflection, have enabled me to bring out, I leave them, with all deference, to the kind judgment of those who consider these pages worth perusing.

On another occasion, perhaps, this subject may be pursued further; upon the present, the limit to space forbids its greater extension.

## APPENDIX.

### *Statistics of the London Truss Society, as published in the Year 1855.*

Males.	Females.		Total.
16,650	687	Left Inguinal, 17,337	} 46,551 Inguinal
28,471	743	Right " 29,214	
315	2,667	Left Femoral, 2,982	
498	3,972	Right " 4,470	} 7,452 Femoral
28,137	366	Double Inguinal	
177	1,795	" Femoral	} 30,475 Double
741	3,064	Umbilical	
229	447	Ventral	} 4,481
1	3	Perineal	
1	4	Obturator	} 5
461	254	Prolapsus Ani	
—	2,517	" Uteri	} 2,816
—	57	" Vaginæ	
—	242	" Vesicæ	
2,713	1,674	have been cured	4,387
78,394	18,492		96,886



Of these 96,886—78,394 were males, and 18,492 were females: 75,054 were cases of inguinal, and 9424 of femoral hernia; or, in per centages, 88·8 per cent. were of the former, and 11·1 of the latter variety.

*Sex.*—Of the inguinal, 97·6 per cent. were males, and 2·3 females; inguinal hernia being most frequent in males by 95·3 per cent.

Of the femoral, 10·5 per cent. were males, and 89·4 females; femoral hernia occurring more frequently in females by 78·9 per cent.

As regards the question of *double hernia*, it will be seen, that of every hundred cases of inguinal, 37·9 per cent. were of this form; and of femoral, 20·9 per cent.

*Side of hernia.*—It is also shown, that single inguinal hernia occurs 62·75 per cent. on the right side, and 37·24 on the left; inguinal hernia being most frequent on the right side by 25·51 per cent. In single femoral hernia, 59·9 per cent. are on the right, and 40 on the left; the difference being in favour of the right, of 19·9 per cent.

ON CONCEALED  
ACCIDENTAL UTERINE HEMORRHAGE.

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BY HENRY OLDHAM, M.D.

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THE following cases illustrate a rare form of hemorrhage, of an accidental kind, where the blood is retained within the womb, little or none escaping externally.

CASE I.—Mrs. K—, æt. 40, a lady in delicate health, engaged me to attend her in her fourth confinement, having suffered seriously from hemorrhage in two of her three former labours, the last of which was unavoidable from placenta prævia. An interval of four years had elapsed since this pregnancy, and the date of her expected labour, which was anticipated in August, 1843. When two months advanced in her present pregnancy, she suffered great mental distress from the loss of her two children from scarlet fever, and since then her mind has been much agitated from this event. At 8 a.m., on the 20th of June, I was desired to see her on account of a slight show of coloured discharge, accompanied with vague abdominal and lumbar pains, resembling both in character and degree the first day of a menstrual time, but with this addition, that she was unusually faint and pale. She was now within six weeks of term, and she was in alarm at the prospect of a premature labour, and from the conviction that she would not survive it. The napkin which was shown me was stained with a palish blood; she was herself pale and tremulous, with a feeble pulse, and generally depressed, but altogether her condition did not appear to me worse than what might be accounted for by the emotional influence of fear on her already debilitated frame. Arrow-root, with brandy, was given her immediately, and five grains of the sesquicarbonate of ammonia, with ʒss. T. Lupul.

in the Inf. Valerian, was ordered every four hours; the strictest rest enjoined, an efficient nurse engaged to watch her, and her alarm was dissipated by words of encouragement. Before 10 p.m. I had seen her three times, and she appeared altogether improved; her spirits were better; the surface warmer; the pulse somewhat stronger; no further discharge, and no uterine pain; but still she had with difficulty been prevented from fainting twice or three times, and she had once vomited. At 6 a.m., on the 21st, I was urgently sent for on account of a more alarming syncope, from which she was rallied with some difficulty; and notwithstanding the liberal use of brandy, she remained in so exhausted a state that I feared she might not recover. My impression was that her symptoms were those of bleeding, but still there was no vaginal discharge; but on passing my hand over the abdomen, I was struck with the increased volume of the uterine tumour—which I had felt before in the morning,—its greater tenseness, and its well-defined outline, and this without uterine action. The os uteri was stretched open to the size of a shilling, but the membranes were flaccid, and the head readily felt and moveable. On reflection I came to the conclusion, that there was internal uterine hemorrhage from placental detachment, and I resolved to rupture the membranes, which was accomplished with a sharp-pointed instrument for the purpose, and a large quantity of liq. amnii drained away. A bandage was placed round the abdomen, and a dose of the Dec. Secale given, which was soon vomited. By the help of various restoratives she revived, and at 9 a.m. labour pains began, the os uteri speedily opened, and before 12 o'clock a dead male child was born. Before the cord was separated, she complained of pain in the region of the uterus, then she became faint, and at the same time blood began to flow freely from the vagina. My hand was promptly introduced within the uterus, which was largely distended, and filled with coagulated and fluid blood; the placenta was loose, and as I withdrew my hand, making pressure with the other over the abdomen, the contents of the uterus were rapidly expelled, and a large quantity of blood was lost. The uterus contracted well, and there was no recurrence of hemorrhage. The extreme exhaustion which followed left this patient for some time in a

perilous state; and although eventually she recovered, for years she may be said to have led a feeble life from the morbid consequences of loss of blood.

The drawing which accompanies this paper is a copy of the placenta, which is preserved in the museum of Guy's Hospital, and affords a valuable illustration of the process by which nature attempts to control this dangerous accident. The moiety of the placenta which was detached is at once recognised by the appearance of a deep excavation, the scalloped edge towards the centre of the placenta being sharply defined, as though it had been cut. There is really no loss of substance whatever, but the placenta has been pressed in and thinned by the collection of coagulated blood between it and the uterine walls; and it is the decidua on the uterine face of the placenta which is seen, on which are marked the furrowed outlines which represent the mould of the collected clots. The steady force which must have been exerted on the placenta to have so completely obliterated its maternal vascular cell-structure, must have been considerable, and it is remarkable that the remaining portion of the placenta should have been able to preserve its adhesion to the uterus in spite of it. (See Plate III.)

*(Reported by C. D. KINGSFORD.)*

CASE II.—At 8 p.m., October 23d, 1850, I was called to see a patient of the Lying-in Charity, Ann W—, æt. 40, residing in the Borough; and found her partially collapsed, the extremities and surface of the body cold, countenance blanched, and no pulse to be felt at the wrist. I immediately gave the patient some brandy, and ordered warm water to the feet, and extra covering. Having instituted a vaginal examination, I found the os uteri about the size of a half-crown, and the head presenting. Upon inquiry I learnt that she had suddenly fainted in the morning, and that syncope had followed after the slightest exertion throughout the day; and that this was her tenth confinement. In the course of an hour warmth returned to the body, and a quick, feeble pulse became perceptible. She had suffered from no external hemorrhage as yet; and being informed that she had passed no water since 10 a.m., a catheter was introduced, and about half a pint of high-coloured



urine drawn off. At 11½ p.m. I received a message that a coloured discharge had appeared. Mr. Morgan, therefore, accompanied me, and we found that a small amount of hemorrhage had taken place, but that labour had not advanced; the abdomen was very much distended, so large as to render it very probable that there were either twins or that dropsy of the amnion existed. In consequence of the hemorrhage, it was deemed advisable to rupture the membranes; no unusual amount of liquor amnii, however, escaped. I remained with the patient until 12¼ o'clock, when she was in good spirits, and her pulse, although still very feeble, yet not so quick. At 2 a.m. I again visited her, and found the os uteri fully dilated, and the head of the child low down in the cavity of the pelvis. The fœtus, however, was dead, no scalp tumour existing, and the bones of the head being easily moved on each other. Although the uterus had thus rapidly dilated, yet she had experienced no regular uterine pains. She seemed to improve in her general symptoms, and took her nourishment well; and wished to get up, as she was tired of lying in one position, but was strictly forbidden. Not a single labour pain occurred up to 3½ a.m., when Tinct. Secale Cornut, ʒj, was administered, in some brandy and water, and repeated without the brandy in half an hour, without, however, producing any effect. At 4½ a.m. she became restless, and being somewhat alarmed at the absence of pain, and no hemorrhage having taken place since the membranes were ruptured, and the patient being apparently in a better condition than during the early part of the evening, it seemed to me highly probable, if an hour's rest could be procured, that pains might come on, and the fœtus be soon expelled, as, from its low position in the pelvis, a very few pains would be sufficient to accomplish delivery. I therefore administered a grain of opium, and left at a quarter to five o'clock, with orders to be sent for immediately any change took place.

At 6 a.m. I was summoned, and found the opium had not acted, and that the patient was becoming exhausted, the pulse more rapid and feeble; the surface of the body was, however, warm, and she spoke to me. A little brandy and water was administered; and having ascertained that no hemorrhage had

occurred, I hastened to Mr. Morgan, and was urging the necessity of delivering as soon as possible by forceps, when I was again sent for, and, on entering the room, found the patient in articulo mortis. It appears that, shortly after I left, the patient attempted to get up in bed, and turned on her back in a fainting state; no hemorrhage had appeared externally, and on Mr. Morgan's arrival (in about ten minutes) with the instruments to deliver, the woman was no more.

When the body was laid out, an hour after death, a large quantity of blood came away, completely saturating the bed and bedding.

*Autopsy, twenty-six hours after death.*—The body was perfectly exsanguine, and on making an incision into the abdominal cavity, the uterus, which was very much enlarged, was seen to occupy the entire space, the transverse colon and portion of the omentum at the fundus of the uterus being the only part of the intestines visible.

A longitudinal incision was made into the uterus, and the greater portion of its interior found filled with large black clots of blood, sufficient in quantity to fill two ordinary-sized chamber-vessels. The fœtus was occupying the left oblique diameter of the uterus, and the placenta found detached from the right lower third of uterus, excepting a small portion only, occupying about an inch and a half in circumference, low down towards the cervix uteri. The fœtus, a male, was unusually large.

CASE III.—Margaret K—, an Irishwoman, æt. 38, when advanced between the eighth and ninth month of her eighth pregnancy, applied at the Lying-in Charity for an attendant, on account of some pains which had come on after drinking and carousing on Christmas-day, 1855. She was seen by the senior obstetric assistant, Mr. Nason, at 4.30 p.m., who found her with the os uteri dilated to the size of a shilling, the head presenting, but without labour pains. She was cold and sweating, but he attributed her condition to the drink which, with the companions in her room, she had indulged in. Arrangements were made for her attendance, but finding that no labour pains

occurred, the gentleman left her, without his attention being attracted to anything beyond her state of intoxication. When summoned to her at 11 p.m., she was dead. There had been no external hemorrhage.

*Post-mortem examination.*—The os uteri was dilated to the size of a crown-piece, and the membranes ruptured. The wall of the uterus was so thin that it was opened by the same incision which divided the abdominal wall. About five pints of coagulated blood were found between the placenta and anterior wall of the uterus, the former being entirely detached. The placenta was taken to the hospital, and a drawing made from it. It was flattened and thinned throughout, but one portion of it more distinctly than the rest; and on this there were some well-marked elevations, like the ridges which would mark the outlines of clots, but not at all in so defined a way as in that of Case 1. The placenta was perfectly healthy.

These two cases, which occurred in the district of the Lying-in Charity over which I preside, are included in the Report which is published in another part of this volume; and one other similar case, attended with a fatal issue, is also there noticed. Three cases, therefore, of this accident, all fatal, have occurred within twenty-one years in our Lying-in Charity, out of the 22,498 deliveries; a fact which sufficiently indicates its rarity and great danger.

This form of internal concealed hemorrhage has been noticed by various accoucheurs—by Bandelocque, Burns, Merriman, Blundell, Ingleby, Johnson of Dublin, Robert Lee, and others,—and the recorded cases attest the obscurity of the earlier symptoms, and the extreme peril of the hemorrhage. The practical difficulty is to recognise the signs of internal hemorrhage before they indicate a fatal exhaustion, and to act promptly and decisively in arresting it. The observations which flow from a consideration of these cases may be thus summarily expressed.

1. The gravid uterus, in a normal state, is not filled to distension by its contents, but there is space enough to allow of so much blood being extravasated from the utero-placental



circulation as to endanger and even to compromise the life of the mother, without any or scarcely any escaping through the os uteri. Such a bleeding is an accidental hemorrhage, with the peculiarity of being internal, or, in other words, concealed within the womb.

2. The effort which nature makes, under favorable circumstances, to limit this bleeding, is to form the clot itself into a powerful compress, interposed between the uterine wall on the one side, and the foetal membranes, with the liquor amnii and foetus, with their opposed uterine wall, on the other. The compression thus exercised will exert so much force on the placenta as to close and obliterate its cell-structure, and probably restrain the regurgitation from the uterine veins. Under unfavorable circumstances, with the recurrence of any disturbing cause, this barrier is shifted or broken, and fresh bleeding occurs.

3. The signs of this accident are marked by the general indications of loss of blood, accompanied by more or less distension, or, it may be, partial bulging of the uterus itself, and the passive opening of the os uteri, from the stretch to which its margin is exposed, in common with the entire cavity of the organ. As in other cases of hemorrhage so in this—labour is suspended, from the general exhaustion which ensues, with the additional source of muscular weakness, from its fibres being unduly stretched.

4. The indications for treatment are to favour the formation of coagula, and sustain the general power. An injunction of paramount importance is to forbid exertion of any kind—to maintain the strictest rest, and to encourage mental tranquillity. If, from the recurrence of faintness and general depression, a further bleeding is suspected, the membranes should be ruptured, and the uterus excited by stimulants, ergot of rye, and a bandage; but should it fail to act, then, according to the absolute acquired dilatation or dilatability of the os uteri, delivery should be accomplished by the forceps or by version. If exhaustion is already profound, then all active



efforts for delivery must be postponed to the urgent necessity of sustaining life, by the freest stimulation, and the transfusion of blood.

5. The prognosis in these cases ought to be very guarded, as the accident is always one of great, and sometimes of sudden danger.

### PLATE III.

Representing the placenta in Dr. Oldham's case of concealed hemorrhage.

The drawing exhibits the uterine surface hollowed out by the pressure of the coagulated blood.

Plate III.







# CASES OF LARDACEOUS DISEASE

AND

SOME ALLIED AFFECTIONS.

WITH REMARKS.

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By SAMUEL WILKS, M.D.

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By placing together a considerable number of examples of the above-named disease, we think a better opportunity is afforded for studying its general character, the class of subjects in which it mostly occurs, and the affinities it has with various other morbid conditions of system. Reports of individual instances of the disease are from time to time brought under notice, and specimens are to be found in various museums; but, at the same time, little regard has been given to its general pathology, or at least to some portions of the subject to which we shall have more especially to direct attention. We intend, therefore, to relate briefly, the cases of this class of affection which have occurred of late at Guy's, and to add to these some account of the specimens which are in our museum, and thus we think an importance will of necessity be gained for them, and more especially a distinctive character for some forms of disease which have been but hitherto little observed.

Some of the latter, which we shall have to notice, are no doubt rare, but the lardaceous is by no means an uncommon affection, although constantly overlooked by those whose duties do not familiarise them with post-mortem appearances. If the spleen and kidney, for example, be the subject of the disease, the unpractised eye will often fail to detect the altera-

tion in structure; and if the liver be the organ affected, it is only an extreme amount of disease which is liable to be recognised. From careful experience, therefore, we can say, that this peculiar affection of the spleen may be easily overlooked (unless the organ has been exposed to the air, or lain some time in water), that the kidney, when the subject of the like disease, may be summarily dismissed as a healthy one, or as an example of morbus Brightii, and that a moderately lardaceous liver may be put aside as a fatty one. The affection of the lymphatic glands, which we shall presently notice, and which appears to bear some close relationship to this form of disease, has not yet (as far as we know) been recognised as a peculiar condition, or deserving of a special name in pathology, although, no doubt, instances of it must be constantly met with.

The name *lardaceous*, which was originally given to the disease of the liver of which we have been speaking, is as expressive as any other, and may be applied also to the similar affections of the spleen and kidney. The term *scrofulous* liver, which has been also used, is not so good; for although the disease is often associated with, or accompanies manifestations of *scrofula*, in other parts of the body, yet this coexistence can by no means be always shown to be present; and therefore a distinctive name for this peculiar visceral change is much to be preferred. There can be doubt that the *strumous* and *lardaceous* conditions are nearly allied, although our early experience caused us to think that this connection was not so close as had been imagined, for in the first well-marked cases of the disease which we witnessed, there were none of the appearances usually known as *scrofulous*, present in the body, except a primary chronic affection of the bone for which general usage had supplied this term. Further observation, however, and the actual concurrence of *lardaceous* disease and tubercles in the same person, proved the intimacy of the two affections. Notwithstanding this, the most important fact in reference to the disease is the one just mentioned, and spoken of by Budd and others, that the extreme examples of this morbid change, especially when it affects the liver, are to be found, not in connection with ordinary tuberculous maladies, but with chronic disease of the bone, especially that form

occurring in young people which has a constitutional origin, and which is generally styled scrofulous. Thus, in children and young persons who have suffered long with necrosis, or ulceration of the spine, hip-joint, &c., and have been bed-ridden for months or years, a tumid condition of the abdomen, with œdema of the legs, is very often seen to ensue, and this particular condition of liver is found after death. More recent observations have also shown that, although the most extreme form of the disease, as first noticed, is met with in the liver, yet that the spleen and kidneys are very commonly affected in a similar manner. In reference to the scrofulous nature of the malady, there is the interesting fact, which will be gathered from the perusal of the cases below mentioned, that in many instances the disease of the bone was not of the kind called scrofulous, or constitutional, but arose from accident, or from acquired maladies, as syphilis or mercurialism. As, however, all chronic affections of this part of the human frame, arising, in the first place, from accident, do not terminate by the development of lardaceous matter in the internal viscera, it might be fairly conjectured that the subjects of the injuries where this result does follow were previously of a scrofulous diathesis; but this requires proof. Our present experience, however, clearly shows that the lardaceous disease is more intimately connected with affections of the osseous system than with any other class of maladies. The close connection between disease of the bone and the affection under consideration, is seen in the fact, that out of 36 cases of lardaceous viscera, 16 had necrosed bone, and 11 very evident disease of a syphilitic or other affection of the osseous system; leaving only 9 where no history existed of disease of the latter description. For the sake of perspicuity, we shall allow this fact to decide the separation of the cases into those which had disease of bone and those which had not. The former again into those where the bone was extensively diseased, as by necrosis, and those where the osseous system as a whole was affected, but without any great local manifestation of mischief. Of those cases which had not disease of the bone we shall distinguish a third class, where the lardaceous condition existed alone, and appeared to be primary; a fourth class where it was associated with phthisis; and a fifth class consisting of cases altogether dif-



ferent from the former, but characterised by a peculiar disease of the spleen and an enlargement of the lymphatic glands, cases mentioned in this place on account of the close alliance, hereafter to be spoken of, between them and the lardaceous affection.

# CLASS I.—CASES OF LARDACEOUS DISEASE CONNECTED WITH NECROSIS AND CARIES OF BONE.

CASE 1.—A young man, aged about 20 years, was admitted into the hospital in 1851, apparently suffering from phthisis or other tubercular disease; there was, however, no malady detected in the chest, but the liver was felt descending very low in the abdomen, and there were chronic sores, exposing the bone on the head. The patient died in a few days, and, on post-mortem examination, the os frontis was found to be much thickened from chronic inflammation; the surface was ulcerated and bathed in pus. Lungs were healthy. Small and large intestines were ulcerated. *Liver* weighed 6½ lb., and presented a good sample of the lardaceous disease; the lobules were distinctly mapped out, and the structure was so firm that the finest transparent section could be made. Towards the surface of the organ some of the lobules were separated by opaque white lines of fat. In the transparent parts the hepatic cells were very numerous, but small, and contained no fat. In shape the liver was completely moulded to the spine and kidneys. Lymphatic system healthy. Kidneys hard, but apparently healthy.

CASE 2.—William H—, æt. 24, was under Dr. Rees' care in January, 1852. Was a painter, and been ill eighteen months with an abscess in the chest, and for which he had been at Margate Infirmary. The last few months he had become very thin. His appetite was often ravenous, but he had no other marked symptoms. He was taken in for phthisis, but it was found that no pulmonary disease existed. The liver was felt to be much enlarged, and there was a sore over the chest. He gradually wasted and sank. The body, though of moderate height, weighed only 57 lb. Three or four sinuses, from which pus was running, existed on the chest, and these led down to diseased bone of the sternum. In the neck there were hard, red cicatrices, as of former strumous ulcers. The liver was clearly seen, through the abdominal walls, reaching to the ilia. The lungs were quite healthy, containing no tubercle. Heart healthy. *Liver* nearly filled the abdomen, weighed 9½ lb., and was moulded to the form of the organs with which it came in contact. It was an extreme example of the lardaceous disease; it contained no fat, and the secreting cells were about half their natural size. *Spleen* about four times its natural size, very firm (lardaceous?). *Kidneys* small and hard (lardaceous?). Intestines, diphtheritic inflammation of mucous membrane. No tubercular disease in any part of the body.

CASE 3.—A little girl, æt. 11. Kept her bed in the hospital for more than a year with disease of the spine and paralysis. The post-mortem examination displayed extensive strumous disease of the cervical and dorsal vertebræ, involving the cord. Lungs full of strumous deposit, and having a large cavity communicating



with the diseased bones of the spine; they contained no miliary tubercle. Heart healthy. *Liver* weighed  $4\frac{1}{2}$  lb., and was in an extreme degree lardaceous. The acini were remarkably well mapped out on the surface. The microscope showed the secreting cells small and wasted. Spleen healthy. Kidneys healthy. Intestines contained a few tubercular ulcers. Mesenteric glands healthy. The legs were cedematous, and the chest and abdomen contained a small quantity of serum.

CASE 4.—Eliza M—, æt. 26, was under Dr. Barlow's care in 1853. Two years before her admission she fell and struck her back and hip; this was followed by hæmaturia and general ill health. A year afterwards she had a premature labour, succeeded by the formation of an abscess over the hip; this continued open, and the urine again became bloody. When admitted she was found to have a fistulous opening over the ilium, leading to diseased bone, and another similar one in the loin. She died at the expiration of eight months with phthisis, but shortly before her death her left shoulder-joint became inflamed and suppurated. On post-mortem examination the lungs were found to be extensively disorganized. *Liver* weighed 6 lb. 3 oz., and was well-marked lardaceous. Left kidney quite destroyed by an old suppuration. Spleen healthy. Intestines contained tubercular ulcers. The bones of the ilium much diseased, and those of the shoulder-joint necrosed.

CASE 5.—George A—, æt. 16, was under Mr. Hilton's care in 1853. He had suffered from a disease of the left elbow-joint ever since infancy, and had been in the hospital with it four years before, and left with it nearly ankylosed. A year before the last admission the right elbow-joint became affected, accompanied by abscesses. When admitted the left elbow-joint was ankylosed, although there were sinuses around it still open; the right was swollen and suppurating. Shortly afterwards a sore was observed on the face, and subsequently some of the malar bone exfoliated. On post-mortem examination the body was seen to be very small and wasted. Brain healthy. Lungs contained strumous deposit and small vomicae. Cervical glands contained a cheesy material. Mesenteric glands enlarged, but no adventitious deposit. *Liver* nearly filled the abdomen, and moulded to its shape; the organ presented an extreme example of lardaceous disease, and weighed 5 lb. 14 oz.

CASE 6.—Joseph P—, æt. 47, under Mr. Hilton in 1854. Three years before, he had pricked the end of his thumb; suppuration took place, and subsequently the bone became affected. Pieces of necrosed bone were at different times removed. Shortly after his final admission to the hospital, with inflammation of the arm, he sank into a comatose state, and gradually died from uræmic poisoning. A post-mortem examination showed the body to be much wasted; the metacarpal bone of the thumb was exposed, and old sores were observable on the legs. Brain healthy. Lungs and heart healthy. Lymphatic glands healthy. *Liver* had undergone a lardaceous and fatty degeneration, the former not very far advanced. *Spleen* very firm and hard, and was a well-marked example of the lardaceous or waxy change. The adventitious material occupying the Malpighian corpuscles as round transparent bodies. *Kidneys* mottled, and contained inflammatory and fatty products in the tubules. The Malpighian bodies also contained some lardaceous matter.

CASE 7.—Anthony B—, æt. 8, was under Mr. Hilton's care, in 1854, for disease of the hip. The boy had had chronic hip disease for two years, and walked with a crutch, but unfortunately falling he fractured the thigh, at the upper part, on the same side. He lay in the hospital for three months; there was no attempt to repair

the injury, and the old sinuses about the joint continued to discharge. He died at last with acute arachnitis. The post-mortem examination showed acute tubercular inflammation of the brain. Lungs contained miliary tubercle and soft strumous deposit. Bronchial glands much enlarged from strumous infiltration. Intestines showed commencing tubercular ulceration. Mesenteric glands affected in the same way as the bronchial, and also the lumbar, extending upwards from the diseased joint. *Liver* lardaceous to a moderate extent. *Spleen* considerably so, containing, as usual, large rounded transparent masses. Kidney contained a few tubercles.

CASE 8.—Ann O—, æt. 47, was under Mr. Cock's care, in 1855, for syphilitic necrosis of the bones of the head and face, and general cachexia. She had suffered from the effects of syphilis and mercury for twenty years. Before her death the ankles and face became œdematous, and the patient sank into a quiet stupor, denoting an impaired action of the kidney. The body was wasted; the nose and palate bones were quite destroyed, nodes on tibiæ, and scars existed on the extremities. Brain healthy. Lungs and heart healthy. *Liver*, surface nodulated and uneven from old capsulitis; the interior showed considerable lardaceous and fatty degeneration, and also a considerable increase of dense fibrous tissue, as in early cirrhosis. *Spleen* was considerably advanced in the lardaceous change, more than half its substance being occupied by the adventitious material. *Kidneys* mottled and fatty; they also contained some transparent lardaceous material.

CASE 9.—William S—, æt. 28, under Mr. Hilton's care, in 1855. He had suffered for a year and a half from chronic inflammation of the knee-joint. He was admitted in a very low state, with suppuration of the joint, in which the head of the tibia was involved. He shortly died of pneumonia. On examination, besides the hepatised lung, the liver was found to be fatty, and the *spleen* extensively lardaceous and enlarged.

CASE 10.—Thomas F—, æt. 40, was under Mr. Birkett's care, in 1855. Twelve years before, he had fallen from a height, and jammed both his legs between some timber. Large ulcers formed, one of which never healed, and at last the bone became affected. He kept his bed for eight months, and at last had the leg removed, the ulcer being considered carcinomatous. The stump never healed, and in a month's time he died. The lungs were found in a recent state of inflammation, and which had been the immediate cause of death. The liver was in a state of advancing cirrhosis. *Spleen* weighed 1 lb. 12 oz., and was extremely lardaceous.

CASE 11.—James D—, æt. 18, was under Mr. Hilton, in 1855, for disease of the hip. Four years before, he had struck his hip while at play; inflammation and suppuration followed, and subsequently he either kept his bed, or walked about on crutches, with a partially ankylosed joint and discharging sinuses. Being worse, he again came to the hospital, and died, after keeping his bed four months. The post-mortem examination showed the body to be extremely wasted and small. The original structures of the joint were entirely destroyed, and some bone was found necrosed. Lungs healthy, with the exception of a few small nodules of granular (strumous?) deposit. Intestines had a small ulcer on the ileo-cæcal valve. Lymphatic glands universally healthy. *Liver* very large, and more than proportionally heavy: presented an extreme example of the lardaceous degeneration, a section of it appearing like wax, consisting of a semi-transparent substance, quite uniform, and presenting no trace of structure, except here and there a small blood-vessel transuding a watery fluid. Towards the circumference, the structure of the

organ was not so far destroyed; the lobules could be distinctly traced out, and white lines of fat were seen running between them. The denser parts of the liver underwent no change by keeping many weeks, and the specific gravity of these was 1084. The weight of the whole organ 6 lb. 14 oz. *Spleen* large and firm, weighing 11 oz., and in a state of advanced lardaceous disease. *Kidneys* presented no remarkable appearance to the naked eye, except being peculiarly firm, white, and tough. The microscope showed, when a thin section was made, the Malpighian corpuscles as round transparent bodies, and where the capsule was torn off the tuft of capillaries was seen covered with the same transparent lardaceous material: the smaller arteries supplying them were also seen surrounded by a similar matter.

CASE 12.—James L—, æt. 27, admitted under Mr. Hilton's care, on July 26th, 1854. He was a carpenter, and seven weeks before had struck his knee with an axe. The wound was slight; but synovitis followed. Abscesses subsequently formed around the joint, the cartilages became destroyed, and ankylosis was commencing to take place. Fresh inflammation, however, further affected the part, and his health suffering, in April 1855 the leg was removed. The recovery was very slow, on account of sloughing and suppuration; but the patient was able to walk about in the autumn, and was convalescent, when, in January of 1856, suppuration occurred in the other leg, and the bone of the pelvis became involved, and he died at last in April of the same year, nearly two years after the receipt of the injury. A few months before his death he had a severe dysenteric attack. The post-mortem examination showed the stump healed; but the hip of the same side partially ankylosed. The ilium of other side was much necrosed; around the dead portions, as well as in the pubis, the bone was so soft that it could be cut with a knife; it was of a red colour, and quite pulpy. The microscope showed no lardaceous matter, but abundance of nucleated exudation cells. *Liver* was not much above the ordinary size, but considerably heavier, weighing 6 lb. It was of a yellowish colour, and presented externally the appearance of the simple fatty organ. A section, however, showed that fat and lardaceous matter were present in nearly equal proportions. The translucent structure was seen pervading the organ in all directions, and amongst it the ordinary fatty-looking hepatic tissue; the latter being more yellow than usual, from considerable biliary engorgement. The whole organ cut with a crispness in the same way as in the ordinary lardaceous disease. Sections displayed by the microscope tolerably defined lobules, surrounded by circles of fat, as in ordinary incipient fat liver, the lobules themselves consisting of this translucent structure. There was none of the opaque or dead-white fatty matter present. *Spleen* very firm, weight 8 oz., presented the disease in its usual form, the Malpighian corpuscles being converted into round transparent bodies. *Kidneys* weighed 15 oz., very firm, white, and tough. Secreting tubules, in parts, contained much granular matter, and the Malpighian tufts showed in a slight degree the presence of lardaceous deposit. Large intestines had the mucous membrane corrugated, and of a slate colour, as if it had been once inflamed. Lymphatic glands healthy. Lungs healthy. No tubercle discoverable in any part.

CASE 13.—Caroline J—, æt. 26, admitted under Dr. Addison, in June, 1856. She was a married woman, with no history of scrofula or consumption in her family. Six years before, she received a kick on her left leg, followed by an ulcer, which persisted ever since; and during the last six months her health had been bad, with a gradually increasing swelling of the legs and abdomen. On admission,



she was seen to be greatly wasted, cachectic looking, with ascites and slight swelling of legs. There was a fungating ulcer on the left tibia, which appeared to be carcinomatous, and which poured forth a watery discharge. Epistaxis was frequent. The urine was scanty and albuminous. She was tapped, with great relief, after which the liver was felt much enlarged, but death took place a fortnight subsequently. *Post-mortem*: No appearance of tubercle in any part of the body. Lungs and heart healthy. Abdominal parietes adherent to liver. *Liver* much enlarged and very heavy; infiltrated throughout with the lardaceous material. A section showed the structure to be peculiar, from being nodulated, giving it somewhat the appearance of a cirrhotic organ, and also from the fact of the peculiar white material, such as has been described in former cases, scattered throughout the organ in distinct masses; weight, 7 lb. 9 oz. *Spleen* large, firm, slightly lardaceous; weight, 10½ oz. *Kidneys*, remarkable specimen of the lardaceous disease, the organs being large, very firm, translucent looking, and feeling like masses of wax. They were also mottled by a white deposit within and without the tubes. Weight, 17 oz. *Mesenteric and lumbar glands* slightly enlarged, white, and very soft. The leg showed no traces of cancer; but the whole tibia was soft and diseased.

CASE 14.—Henry H—, æt. 42, under Mr. Cock, in July, 1856. About four years before his death he began to complain of pain in his back. This continued some months, when an abscess formed in his buttock. At the end of the year 1855, psoas abscesses appeared, which shortly after broke, and continued to discharge until his death. *Post-mortem*: Body extremely wasted. Extensive necrosis of lower lumbar vertebræ, sacrum, and adjoining ilia. *Liver* small, but very heavy; the cut surface glistened, as in the lardaceous organ, but the ordinary structure was still apparent. The crispness on cutting it was quite peculiar to the disease, although this was not sufficiently far advanced to form any distinct white masses of pure lardaceous matter. *Spleen* slightly affected by the same disease. A few of the mesenteric, lumbar, and bronchial glands contained a small quantity of strumous matter. Lungs healthy; no tubercle.

Among the old specimens of lardaceous liver preserved in our museum, only three have histories attached to them. Two were connected with disease of the bones, and the cases are as follows:

CASE 15.—James M—, æt. 5, was under Mr. Morgan's care, in 1827, for disease of the spine. He was confined to his bed two years, and his back was seared by abscesses and sinuses, leading to necrosed bone. The abdomen was very large from the increased size of the liver. Upon post-mortem examination, the *liver* was found much enlarged, and weighed 3 lb. 6 oz., very firm, and looking like an undressed ham. Lungs healthy. Intestines healthy. Mesenteric glands much enlarged, but free from tubercle. Spleen healthy. Kidneys large, and texture very firm (lardaceous?).

CASE 16.—Joseph R—, æt. 10, under Mr. Key's care, in 1832, for chronic disease of the hip-joint, attended with suppuration and dislocation. The boy had been ill more than two years. On post-mortem examination, the *liver* was found very large and pale, also firm and solid, and the secretion pale. Spleen contained granules



(lardaceous?). Kidneys were of a yellowish, opaque, white texture (lardaceous?). Mesenteric glands large and firm. The whole of head of femur was found necrosed.

## CLASS II.—CASES OF LARDACEOUS DISEASE CONNECTED WITH SYPHILIS, RHEUMATISM, &c.

CASE 17.—Thomas M—, æt. 15, under Dr. Barlow, in 1848. Three years ago he received a severe injury to the thigh, affecting the bone, and health not good since. For some months dyspnœa, cough, and dropsy. No history of rheumatism. Admitted for disease of the heart, and dropsy. The post-mortem inspection showed a diseased mitral valve. Lungs healthy. Liver much enlarged, reaching below the ribs, and having the ordinary lardaceous appearance, and between the lobules there appeared an excess of fibrous tissue.

CASE 18.—William J—, æt. 10, in hospital in 1852. Had scarlatina, and some obscure history of rheumatism, five years before. Admitted for albuminuria. On post-mortem examination, the pericardium was found adherent and the mitral diseased. Lungs apoplectic, otherwise healthy. Kidneys large, white, and mottled. Liver large and lardaceous, and towards the circumference an opaque white matter streaking the transparent tissue.

CASE 19.—Thomas M—, æt. 28, admitted under Dr. Barlow, in 1853. For a considerable time suffered from syphilitic pains in the bones, and for ten months with symptoms of renal disease. Well-marked symptoms of the latter affection existed during another six months in which he was in the hospital. On post-mortem examination the lungs and heart were found healthy. Liver, capsule much thickened, and contracted from old inflammation. This contraction continued into the structure of the liver, producing an advancing cirrhosis. In the lobules, also, there was a transparent substance, which under the microscope could be isolated in distinct pieces, as in the ordinary lardaceous liver. Kidneys rather under the usual size. A section showed the organ to consist of a whitish semitransparent substance, surface smooth, presenting no appearance of deposit or granulations. The microscope showed the natural structure much wasted, and an additional amount of fibrous tissue, accompanied by transparent waxy matter. Intestine healthy. Brain healthy.

CASE 20.—William H—, æt. 34, under Mr. Birkett, in 1854, for stricture. He had been much affected by syphilis, and was in a very cachectic condition. Died from inanition. The post-mortem examination showed the heart and lungs healthy. The liver weighed 4 lb. 10 oz., was in an early condition of cirrhosis, a section presenting well-marked nodules in many parts, and between them much fibrous tissue. The lobules also contained much transparent matter, although the organ was not an extreme example of the lardaceous change. Spleen weighed 1 lb. 7 oz., hard, and contained a number of the usual round, waxy granulations. Kidneys weighed 13 oz., firm, white, fibrous, and waxy. The only approach to strumous deposit in this case was a mass of unorganized lymph found in the testes.

CASE 21.—William H—; æt. 15, under Dr. Rees, in 1854. He had only just recovered from rheumatism, and was in a very cachectic condition. Lungs contained tubercle. Bronchial glands also tubercular. Mesenteric glands enlarged. Intestine ulcerated. Liver fatty. Spleen extremely affected by the lardaceous deposit.

CASE 22.—George C—, æt. 37, under Mr. Hilton, in 1854. He had been an hospital patient at different times, during two years, for syphilis, testitis, nodes on the tibiæ, synovitis of the knees, and for general cachexia, when the liver and spleen were found to be much enlarged. He died, at last, from acute pneumonia. Body wasted. Nodes on both legs, one of which being cut into, showed the bone below becoming necrosed. Lungs hepatized, no tubercle. *Liver* much enlarged, weighing 6 lb. 13 oz., showed distinct nodules, as in cirrhosis, and also lardaceous deposit. *Spleen* weighed 2 lb. 5 oz., its structure presenting nothing unnatural. Kidneys healthy. Testes contained masses of degenerate lymph.

CASE 23.—George H—, æt. 24, admitted under Dr. Gull, in 1855. Had suffered from syphilis during a period of five years, and ten months before, broke his arm. His health had been bad since this time, and the arm had been very painful. Very cachectic, knee swollen, ulcer in throat, cough, urine high-coloured and albuminous. He died in a half-comatose condition. Post-mortem examination showed the body wasted, nodes on both tibiæ, legs slightly anasarctous. Lungs contained masses of strumo-pneumonic deposit. Intestines contained no tubercular ulcers. Mesenteric glands enlarged. *Liver* much enlarged, and considerably advanced in the lardaceous degeneration; it also contained an excess of fat. *Spleen* contained round, transparent, lardaceous nodules. *Kidneys* above average size, of firm consistence, no mottling, but contained a considerable amount of fat in the tubes, and lardaceous deposit.

CASE 24.—Alice B—, æt. 29, under Dr. Hughes's care, in 1855. She had had rheumatism and disease of the heart, but admitted for pelvic cellulitis after parturition. The post-mortem examination showed extensive suppuration about the pelvic viscera. Heart had diseased mitral and aortic valves. *Liver* healthy. *Spleen* filled with round, transparent, lardaceous bodies. Kidneys large, white, and mottled.

CASE 25.—William B—, æt. 33, under Dr. Hughes, in 1855. Had had acute rheumatism and syphilis, twice lately. His last attack laid him up six months. Admitted for dropsy, albuminous urine, and bronchitis. The post-mortem examination discovered the lungs recently hepatized. *Liver* in an early stage of cirrhosis. *Spleen* weighed 14½ oz., and was full of lardaceous nodules. *Kidneys* weighed 13 oz. They were of a white colour, and contained some specks of granular and fatty matter; the Malpighian bodies were affected in an extreme degree by lardaceous deposit, appearing as glistening transparent bodies in the field of the microscope.

The following case refers to the only other preparation of lardaceous liver in the museum which has a history :

CASE 26.—William H—, æt. 14, a patient of Dr. Cholmeley, in 1827. At ten years of age he had rheumatism, and which continued off and on several months, when he was taken with palpitation and dropsy. After admission, the swelling of the legs decreased, but the enlargement of the abdomen continued. The urine was coagulable. He was mercurialized without effect. He afterwards had another rheumatic attack, and died of acute pericarditis. Post-mortem examination showed recent inflammation of the heart. Kidneys displayed the mottling degeneration (case mentioned in Bright's Med. Reports). *Liver* reached over to the left side and down to the ilium, was of a light-yellow colour, firm and translucent, much harder than fat liver, but burned when put into the fire.

The most marked specimen of waxy spleen among the old preparations in the museum has no history. Another specimen has the following:

CASE 27.—William B—, æt. 30, under Mr. Morgan, in 1830, for scrofulous ulcers in the axilla and neck, accompanied by general cachexia. Had long been in the syphilitic ward, and had taken large quantities of mercury. About four months before his death abdominal dropsy made its appearance.

### CLASS III.—CASES OF SIMPLE LARDACEOUS DISEASE.

It will be seen that we have only two cases where the lardaceous disease occurred alone, and seemed to be the primary affection; but it is possible that, even here, the absence of a history of syphilitic or strumous disease of bone, may be due to an oversight on the part of the observer.

CASE 28.—Stephen F—, æt. 51, under Dr. Hughes, in 1855. He had had various dropsical attacks during a period of eight years, and been in different hospitals. On admission he was generally though moderately dropsical, and urine albuminous. After a short time he died. The heart was found hypertrophied. Liver structurally healthy. *Spleen* very hard and lardaceous, presenting the usual appearance of this disease. *Kidneys* of usual size and weight. Colour whitish, and transparent-looking. The microscope showed considerable lardaceous change in the organ, the Malpighian bodies presenting a very remarkable appearance under the microscope as transparent glistening bodies. There was an amorphous exudation in the tubes, and the renal arteries were diseased.

CASE 29.—Mary Ann S—, æt. 9½ years, was a patient of Dr. Wilks, at the Surrey Dispensary, in August, 1852. She was only seen once before her death, and therefore there was not much opportunity to take note of any particular symptoms. Her mother stated that she was in good health until three weeks before, when she became irritable and very drowsy, constantly falling to sleep; at the end of a week she began to complain of pain in her abdomen, and at the same time her legs and face became swollen. On Dr. Wilks's visit, he found the child moderately but generally anasarcaous, and with fluid in the chest and abdomen. The urine was examined by the house-surgeon, and said to be healthy. On the following day the child died, and a post-mortem examination was made. There was general passive dropsy, both within as well as outside the body. At first sight all the organs appeared healthy, but upon incising the liver it was found to have undergone the lardaceous degeneration, being of the usual transparent hue and exuding a watery blood. The kidneys appeared quite healthy. It is to be regretted that there was great difficulty in removing any parts of the organs, as it would have been more satisfactory to have subjected the kidneys to a microscopic examination.



#### CLASS IV.—CASES OF LARDACEOUS DISEASE OCCURRING WITH PHTHISIS.

CASE 30.—Thomas C—, æt. 21, under Dr. Rees, in 1853. Seven months before his death he broke his ribs; the accident was followed by hæmoptysis, and shortly afterwards by all the symptoms of phthisis. The latter disease ran its usual course. The post-mortem examination showed the lungs much disorganized. *Liver* fatty and lardaceous, larger and heavier than usual, emitting a watery blood, and having all the usual characters of this disease. *Spleen* healthy. *Kidneys* appeared healthy to the eye, but the microscope showed some adventitious lardaceous matter present in them. Mesenteric glands enlarged, and intestines had tubercular ulceration. There was no disease apparent about the injured ribs.

CASE 31.—Charlotte E—, under Dr. Barlow, in 1854. Had chest disease two years, and abdomen enlarged eight months. Body much wasted. Lungs extensively disorganized. Fluid in abdomen. *Liver* extremely lardaceous, and a considerable amount of white opaque fatty matter running between the lobules; weight  $7\frac{1}{2}$  lb. *Spleen* slightly affected in a similar way.

CASE 32.—George B—, æt. 20, under Dr. Hughes, in 1854, with phthisis and albuminuria. After death the lung found much disorganized. Bronchial glands contained tubercular matter. *Spleen* hard, solid, and lardaceous. *Liver* fatty. *Kidneys* mottled, containing the ordinary exudation in tubes, with some lardaceous matter.

CASE 33.—Frederick S—, æt. 18, under Dr. Addison, in 1855, for phthisis. The post-mortem examination showed extensive disorganization of the lungs. Lymphatic glands in various parts contained strumous matter. *Liver* fatty and lardaceous. *Spleen* was an extreme example of the lardaceous organ; two supernumerary spleens were affected in like manner. *Kidneys* healthy. Intestines were subject of tubercular ulceration.

CASE 34.—Thomas A—, æt. 40, under Dr. Hughes, in 1856, and died soon after admission. He was a sailor; there was no history. He had deep scars on his legs. Lungs disorganized. *Liver* fatty. *Spleen* had undergone the lardaceous change. Intestine, tubercular ulceration.

CASE 35.—William W—, æt. 38, under Dr. Addison, in 1856. He was a smith, and four years before had suffered from rheumatism; he had not been well since this time. Phthisical symptoms had existed seven months. Post-mortem examination: Lungs extremely disorganized. Intestines were subject of tubercular ulceration. *Liver* fatty. *Spleen*, 11 oz. in weight, lardaceous.

#### CLASS V.—CASES OF A PECULIAR ENLARGEMENT OF THE LYMPHATIC GLANDS FREQUENTLY ASSOCIATED WITH DISEASE OF THE SPLEEN.

The following ten cases present a peculiar affection of the lymphatic glands. The first four (A) are seen to be connected



with lardaceous and tuberculous affections: the last six (B) with a peculiar disease of the spleen, and appear to constitute a special form of malady.

A. *Enlargement of Lymphatic Glands combined with Lardaceous, Tuberculous Disease, &c.*

CASE 36.—Joseph L—, æt. 23, under Dr. Gull, in 1854. When a child had strumous abscesses in the neck, the cicatrices of which were still visible. Been ailing six months before admission with general debility, and for six weeks he had observed his abdomen swell. Noticed lumps in the groin for eight years. On admission he was extremely ill and emaciated. Abdomen distended, and liver felt much enlarged. Legs anasarcaous. Glands enlarged into distinct tumours in the right groin. The patient gradually sank. On post-mortem examination the body was seen to be extremely wasted. Lungs healthy. *Liver* of immense size, and almost filling the abdomen; it weighed 14 lb., and its structure was of the extreme lardaceous kind, a section showing for the most part a uniform white albuminous appearance, and parts having white lines of fat interspersed. *Spleen* weighed 1 lb., and was also lardaceous; besides presenting the usual appearance of distinct nodules of the adventitious material, there were striæ and masses of the same running throughout the substance. *Lumbar glands* very much enlarged, many the size of hen's eggs; they appeared to the eye as if simply hypertrophied; they were firm, and had rather a transparent or gelatinous appearance. Some few contained in their interior a dead yellow lymph (strumous?). The glands in the groin were structurally the same. *Kidneys* of natural size, dense, white, and transparent looking; microscope showed an abundance of adventitious fibrous tissue and waxy material in the secreting portion.

The following case refers to an old preparation in the museum, which appears to be lost, but the history will be found in vol. iii, 1st series of the 'Guy's Hospital Reports.'

CASE 37.—Joseph P—, æt. 10, admitted into the hospital in 1830, with enlargement of the abdomen, from a tumour, which could be felt reaching as low as the pelvis. This had been coming eighteen months. At the end of another six months he was in an extreme state of emaciation, and a row of enlarged glands had appeared beneath the jaw. The body was very wasted. *Liver* weighed 11 lb., flat and smooth, and when divided resembled the cut ends of bundles of muscular fibre. *Kidney* large. *Spleen* at least six times its natural size, solid, and pretty thickly studded with very small light bodies. The whole of *lumbar glands* were greatly enlarged, of fleshy consistence, and homogeneous structure, with nothing of the fungoid or scrofulous appearance; some red from ecchymosis, but most of them flesh-colour, with a yellow tinge.

CASE 38.—William R—, æt. 37, under Dr. Hughes, in 1856. The patient was exceedingly ill, and therefore the history he gave was not to be relied on. He stated that he was a steward on board ship, and had left the East three months, and was well until his arrival in England, five weeks before admission, although he

afterwards said he was unwell during his voyage home. He had never had ague or dysentery. When admitted he was in a typhoid state; left leg anasarca; liver to be felt below the ribs. On the following day his skin became yellow, and jaundice rapidly ensued. After four days more he died. Post-mortem: Larynx ulcerated. Lungs contained tubercles; parts inflamed and in a state of purulent infiltration. *Bronchial glands* were much enlarged; a mass of them the size of an egg existed at the bifurcation of the trachea; and a small mass of degenerated lymph existed in the midst of one. The structure of the glands generally was firm and transparent, like those in the abdomen. Intestine, tubercular ulceration. *Mesenteric glands* about three times their natural size, and having the same appearance as the other absorbent glands. *Liver* was filled with a number of small yellow tubercular deposits, each about the size of a pin's head; these, when minutely examined, appeared to run in the course of the portal vessels, and surrounded the minute terminations of the vessels and ducts; under the microscope they presented not only amorphous or imperfect cells, as in tubercles, but some a firm fibrous tissue displaying nuclei by addition of acetic acid; and also some semi-transparent flakes of an albuminous material. Around the biliary ducts were some enlarged lymphatic glands, resembling those before mentioned. Weight of liver, 6 lb. 9 oz. *Spleen* very large and hard; upon incising it a large abscess was opened; it was not circumscribed, but the purulent matter infiltrated the surrounding structure. In the remaining parts of the organ a peculiar transparent-looking material, like the melted tallow mentioned in other cases, was seen running throughout its structure; this substance had no particular form, but infiltrated the organ in long masses. When examined more closely it was seen to consist of amorphous transparent lardaceous material, intermixed with a nucleated fibre tissue; besides this substance the spleen contained a vast number of tubercles, resembling those in the liver. Around the vessels at the hilum was a large cluster of hypertrophied glands. Weight 2 lb. 2 oz., after the pus was evacuated. *Lymphatic glands* throughout the body were very much enlarged, and, in addition to those above mentioned, the lower lumbar were much affected; they all resembled one another, were very firm and tough, although a section gave the idea of their being soft: this arose from their transparent and gelatinous appearance. They consisted of a nucleated, fibrillated structure, such as is seen in many fibro-plastic tumours which are removed by the surgeon, and in parts there was the addition of a transparent hyaline substance similar to that in the spleen. Many of the cells were not distinguishable from the ordinary nuclei of the gland. Kidneys healthy. (In addition to the appearances which we wish to represent, there will be seen splenic abscess and pneumonia. Whether the former arose from the simple suppuration of the adventitious deposit, or whether it arose from any other cause (as tropical influence), or whether pyæmic, is not very clear.)

This case is taken from our museum records in connection with the preparation of the enlarged lymphatic glands.

CASE 39.—Thomas B—, æt. 50, under Dr. Bright, in 1830. Came into hospital with large tumours in the neck and groin, which had been growing two years. After death, upon removing the sternum a number of very large glands were seen in the mediastinum, extending to the axilla and neck; the same also in the posterior mediastinum. Ascites considerable. Liver uneven and nodulated (cirrhotic?). A

large mass of tumours, formed by enlarged glands, surrounded the aorta in the abdomen. The glands or tumours all resembled one another, some being as large as eggs. Texture uniform, pale, and slightly transparent. No disposition to suppurate or soften. Some, when first removed, appeared of cartilaginous hardness, but afterwards became softer. Spleen healthy.

*B. Enlargement of the Lymphatic Glands combined with a Peculiar Disease of the Spleen.*

CASE 40.—Lewis P—, æt. 24, under Dr. Hughes, in 1856. He was a blacksmith, living at Lambeth. Habits rather intemperate. His health was good until the middle of December, 1855, when, after being exposed to wet, he was seized with a severe cold, attended by shivering, pain in the back, &c. He did not get rid of this attack for some time, and he was much troubled by daily perspirations. His account led to the belief that he must have had intermittent fever. He never got well, although he went to his work. He soon found himself becoming thinner, but had no cough. On admission he was in a very debilitated condition, so that he was obliged to keep his bed, and his skin was remarkably pale; he also had dyspnœa, and felt so feeble that it seemed a trouble to him to speak. His appearance was that of chlorosis. The spleen could be felt below the ribs. The blood, when examined microscopically, exhibited no marked excess of white corpuscles. Cod-liver oil, iron, wine, and other means were adopted to restore his strength, but without avail, for he gradually became weaker, and he died in April, 1856. The only symptom of organic disease discoverable was a dulness and crepitation at the apex of one lung. Upon post-mortem examination the body was seen to be spare, but not much emaciated. Lungs, both apices indurated by chronic inflammatory and tubercular deposit, and the right containing a very small vomica. There was serum in all the cavities of the body, and the lungs were œdematous. *Lymphatic glands*: These were very much enlarged; those principally affected were the lumbar and posterior mediastinal, which formed a continuous chain of tumours along the whole length of the spine upon each side of the aorta. Their size was not equal to that sometimes seen, the largest not exceeding that of a walnut. A few enlarged glands were also found around the vessels entering the liver and spleen, but none of the cervical, inguinal, or external glands were at all affected, nor the mesenteric. These bodies, when examined, were found to contain no deposit, but their substance was translucent and gelatinous-looking; the smaller were soft, but the larger more than usually tough. They contained, besides their normal structure, much fibrous tissue, and large, ill-formed granule cells. *Spleen* weighed 1 lb. 9 oz. About half of its substance was occupied by opaque white deposits scattered through it; these were masses of a firm, cheesy consistence, possessed of no visible organization, and such as to the naked eye would appear as tubercular. The microscope showed that they contained large cells with granules, scattered through a delicate fibre tissue. Liver and kidneys healthy.

The following four cases are connected with preparations in the museum:

CASE 41.—Joseph S—, æt. 9, under Mr. Morgan, in 1826, for a large ulcer on the scrotum, caused by a puncture to evacuate serum. He had been sleeping with a



brother who died of phthisis, and he had been ailing nine months with pain and increase of size of the abdomen. He died of dropsy. The post-mortem examination showed that the lungs contained a few tubercles at apices. Extensive recent peritonitis, with effusion. Intestines healthy. *Spleen* large, and contained a number of white bodies, of irregular ovoid shape. Kidneys mottled. *Lumbar glands* much enlarged, and accompanying the aorta along the spine as far as the iliac vessels and pelvis. *Mesenteric glands* in like manner enlarged, one or two equalled in size a pigeon's egg, of semi-cartilaginous hardness, and streaked with black matter. Bronchial glands similarly affected. Liver, substance natural, but pervaded by a few tubercles, somewhat larger than peas, and of semicartilaginous hardness. (The spleen, in the catalogue, is said to be pervaded by malignant matter.)

CASE 42.—E. K—, a boy, æt. 10, was under Dr. Bright, in 1828. In good health until thirteen months before, when his health began to fail. A tumour was felt in the right hypochondrium, and the glands in the neck became enlarged. His complexion was pale and wax-like. He lived several weeks longer. After death the *cervical glands* were seen as smooth ovoid masses, connected together by loose cellular tissue. When cut into they appeared almost of cartilaginous consistence, of light colour, slightly vascular, but with no appearance of softening or suppuration. The mediastinal and bronchial glands similarly enlarged. Mesenteric glands slightly enlarged. Lumbar glands much enlarged, as those in the neck. Liver healthy. *Spleen* four times its natural size. Structure altered throughout. When a section was made, at least three fourths were seen to consist of a white opaque matter, almost like tallow, pervading every part, and assuming irregular ovoid and spherical masses, very much as if tallow in a melted state had been injected into the cells of the spleen and then cooled. The glands around the roots of the vessels were all enlarged and hard. (Preparation of spleen, considered to be affected with malignant matter.)

CASE 43.—Samuel W—, æt. 16, under Mr. Key, in 1835. Ill two years, with enlarged glands in neck, and discharging sinuses. He gradually wasted. Body pale, and devoid of fat. *Lymphatic glands* much enlarged, and besides those felt during life in the neck and groin, those about the great vessels in the chest and abdomen were similarly affected. These glands were of a reddish colour, and in parts semi-transparent. Some were very large, and some contained cretaceous deposit. Lungs contained a few tubercles. *Bronchial glands* partook of the general affection of the lymphatic system. Liver large, solid, and pale, and appeared to have a tubercular deposit throughout, in the shape of small whitish bodies, about the size of pins' heads. *Spleen* rather fleshy, and contained a few minute tubercles. Kidneys hard, and contained also a few tubercles. Mesenteric glands slightly hypertrophied. Intestines ulcerated.

CASE 44.—Thomas W—, æt. 50, under Dr. Addison, in 1830. Very pale and cachectic, and the most marked feature in the case was the great enlargement of the absorbent glands in neck, axilla, and groin. Some of these were the size of a pigeon's egg, and a few larger and smaller. After death, the *glands* were found loose, and free from morbid deposit; smooth, whitish, with few blood-vessels. They possessed a slight translucent structure, quite uniform, and exhibiting no trace of softening or suppuration. Appeared to consist of a morbid hypertrophy rather than an adventitious deposit. Glands in groin similarly affected. Lungs and heart healthy. A large mass of glands around Glisson's capsule and along the aorta.



Mesenteric glands slightly affected. Liver large, pale, and slightly granular. *Spleen* much enlarged, and texture firm, and contained an infinite number of small, white, nearly opaque spots, which were thought not to be tubercles.

The only case I will quote from foreign sources shall be the following, recorded by Dr. Markham in the fourth volume of the 'Transactions of the Pathological Society of London,' page 177 :

CASE 45.—A man, æt. 30, had been a patient of Dr. Sibson, in St. Mary's Hospital. He had enjoyed good health up to a period of about a month before admission, and he died about six weeks afterwards. He was very pale, ancles œdematous, abdomen somewhat swollen, urine albuminous, and, from the circumstance of an enlarged gland existing in the neck, it was thought probable that some malignant disease existed internally. At last the man became more wasted, comatose, and jaundiced. The autopsy showed the presence of a cluster of *enlarged glands* in the anterior mediastinum, encircling the aortic arch, and extending down the posterior mediastinum. They were whitish-yellow, hard, and unyielding, resembling medullary carcinoma. The *spleen* contained straw-coloured masses, some as large as a filbert, others as mere specks, distributed through every part of it. Weight, 1 lb. 10½ oz. Similar deposits were found in some of the *mesenteric glands*. Dr. Markham adds : " The appearance of the tumours, and the history of the man's disease, left no doubt as to their malignant character, but when portions of them were tested by the microscope, they were found to contain none of the elements usually given as characteristic of cancerous disease ; they seemed to be simply fibrinous deposits, containing granular matter with granular nuclei, but no trace of a composite cell or of fibroid deposit ; the specimen was carefully examined by Dr. Handfield Jones and Dr. Sieveking, as well as by myself. Some of the proper elements of the spleen were found mingled with the deposits in that organ." Dr. Bristowe was asked further to examine the specimen, and stated that the character assigned by Dr. Markham to the diseased condition of the spleen and lymphatic glands was apparently correct ; there was little evidence to show that they were malignant, and none stronger that they were scrofulous. Dr. Bristowe adds, that the glands were of a grayish fibrous material with opaque yellow circumscribed deposits. The fibrous substance had something of the appearance of scirrhus, but was scarcely so firm, nor did it yield a creamy juice. The yellow deposit was not very unlike tubercle, and consisted of aggregated cells and granular matter disseminated through a delicate fibrous reticulum. The cells of irregular shape, and, with these, oil-globules and cholesterine. The remaining portions of the glands consisted of fibrous tissue. The adventitious matter in the spleen was also fibrinous. .

The first question for inquiry is, what is the nature of this lardaceous disease, and how does it affect the organs which are the subject of it? Pathological investigations of late years have shown that the textures of the body may undergo a variety of changes or degenerations, and thus, as formerly every process was called inflammatory and every change the

result of inflammation, so now we recognise the replacement of healthy tissue by fat, fibre (besides into the previously well-known cancer and tubercle), and many other products, amongst which is the lardaceous material, a principle which appears to be altogether peculiar, and one formerly much overlooked. There are, no doubt, also various other materials produced in the different morbid processes which are so constantly being set up in the body, and it may be that those found in the lymphatic glands and spleen, presently to be mentioned, are of a peculiar kind.

The name lardaceous takes its origin from the resemblance which the liver has, when thus affected, to bacon rind. The cut surface of the organ has a semitransparent appearance, presenting no structure, and feeling, when incised, like a piece of wax, or of wax and lard combined. It can thus be cut into portions of the most regular shape, with the sharpest angles and smoothest surfaces; the thinnest slice can also be taken off by the scalpel for microscopic purposes. There is little change produced in it by water or alcohol, and acids and alkalies do not effect any great alteration in it. It is thus remarkably inert, and may be kept for a great length of time without any signs of decomposition. As regards the nature of the lardaceous substance, it is not composed of fat, nor is it wax or gelatine, or any of the ordinarily well-known animal substances, but an albuminous compound, altogether differing from these, and therefore quite deserving of a new name. When affecting organs in too slight a degree to be recognisable by the naked eye, it is seen by the microscope in the form of rounded or oval masses like horn, presenting no structure, and quite unaffected by the application of ordinary reagents.

The very lax expression *colloid* has been used by some pathologists to designate a transparent viscid fluid or solid substance, found under a great variety of circumstances in all parts of the body, including the disease of which we are speaking. Nothing, however, but confusion can result from making use of the same name to designate the gelatinous-like matter contained in the so-called colloid cancer, the fluid found in cystic disease of the ovary, the material of numerous cysts in other parts of the body, as in the thyroid gland, and

the lardaceous disease now under consideration. The term, however, has even had a much wider application than this, and has been used to designate all those amorphous bodies detected by the microscope in almost every degenerated structure of the human frame, such as the corpora amylacea of the brain. If the same chemical composition be discovered in all these substances, and under all these different conditions, then it will be time enough to make use of a common expression for their designation; but, at present, the circumstances under which they are found, including their whole clinical history, are so different, that no advantage appears to be gained by using so ill-defined an expression as colloid to include them all. The fact is, that there is scarcely an organ in the body, under certain conditions of disease, but which will exhibit amorphous, vitreous-looking masses, by the microscope, and which cannot be distinguished from the ordinary lardaceous material when small quantities of it are examined at a time. To say, however, that the two are alike, would be almost equivalent to declaring that tissues undergoing degeneration form within themselves, among other changes, a certain peculiar albuminous product, which, if occurring in excess, is no other than the well-marked lardaceous material. This may be true, but is not yet proved; and there are many reasons against adopting such an opinion—the lardaceous change being one analogous to the cancerous or tuberculous, whereas the other condition, so frequently found in all organs, is simply a degeneration, as is proved by the presence of cholesterine and other substances of recognised low or deficient organization. In a wasted eye which we lately examined, the contents consisted mainly of a number of these bodies called colloid and cholesterine. These may be also found in the kidney, and particularly in the brain and spinal cord, when long atrophied. In cancer, especially in the epithelial form, they are found in abundance; and apparently similar bodies are occasionally met with in the blood, as described by Gulliver and Hassall. In wasted nervous structures, even another material has been mentioned under the same name, the so-called corpora amylacea. These, probably, have been originally the colloid bodies resulting from the formation of new products in a decaying brain, and then undergoing a further change, until



they are capable of being coloured blue by iodine. It may be as well to state that iodine does not effect this change on the ordinary colloid or lardaceous material, although these substances very readily absorb the test, and thus become coloured by it. They appear of a bright cherry-red colour, while the ordinary tissue retains its original pale hue. From many observations, we can say, that ordinary lardaceous or colloid matter is not turned blue by iodine and sulphuric acid, as some appear to suppose. With regard to the effect of these reagents upon the so-called corpora amylacea, this is a point foreign to our subject; but we may state that the true nature of these latter bodies is not yet satisfactorily shown, whether they be really starch, lignine, or other allied material.

*Lardaceous liver.*—In its extreme form the peculiarity of this disease is very great. The increase of size of the organ is immediately recognised, and, when removed and handled, its more than corresponding density is very remarkable. A liver which at first sight appears to be simply a fat one, may often be known to be pervaded by lardaceous matter by its excessive weight, even before a section is made of it; and this is easily intelligible, when it is stated that the fatty liver sometimes attains as low a specific gravity as 1005, whereas the lardaceous may reach as high as 1085. The sensation upon handling a liver pervaded by this material is very different from that produced by the softness and elasticity of ordinary healthy viscera, being more like that which is experienced in feeling a lump of wax. Its resemblance to this substance is increased by the fact of the organ being moulded to the form of the other viscera with which it comes in contact, having its sides straight, its front flat, and its under surface shapen hollow, to correspond to the kidneys and spleen. When cut, however, the knife passes more readily through than it would through wax, the lardaceous substance being crisper than the latter; and this has suggested its resemblance rather to a turnip. A turnip, on the other hand, is too hard, and therefore, probably, a material imagined to be equal in consistence to these two substances combined would be nearer the truth. In well-marked instances of the disease, the solidity and firmness of the organ is very remarkable, so that the thinnest slice can easily be cut by the scalpel for microscopical pur-



poses, and pieces of all shapes, presenting the acutest angles and sharpest edges, can be readily removed. The appearance to the eye is something like the bacon rind, from which it derives its name, being too translucent for wax, except the latter be seen in a melted state. If the disease be far advanced, the organ will present in its interior no trace of structure, except here and there a blood-vessel, emitting a pale watery blood, the adventitious material presenting merely a uniform smooth surface. In a less degree of disease, such as is generally found towards the circumference of the viscus, the lobules are mapped out in a remarkably clear and defined manner; in fact, in no disease of the liver is the appearance of lobules (which, as a rule, are not marked) so distinct as in this particular morbid condition. This is caused by the lardaceous material being deposited within the lobule, in and among the secreting cells, causing it to appear as a distinct transparent body, and made more definite, often, by a slight fatty degeneration of its margin; for this fat, being mixed with the lardaceous matter, produces an opaque white material, which passes completely around and amongst the lobules, mapping them out in the most perfect manner. Towards the edges of a lardaceous liver, then, this appearance is generally seen—a dead white opaque matter running in the course of the portal vessels, and between these the transparent lobules themselves, with the hepatic vein in their centre. Fat is no essential element of the disease, for the most extreme instances do not contain it, except it may be towards the circumference of the organ, as already stated. In cases of phthisis, however, we often see the two coexisting: the liver may appear to the eye only yellow and fatty, but at the same time remarkably heavy; and when a section is made, the structure will be seen to be composed of the two morbid conditions, in various proportions. Probably, towards the circumference the organ will be yellow, soft, and altogether fatty, while in the centre the same appearance will be seen, but pervading it there will be the ordinary-looking, translucent, firm lardaceous matter. By the microscope, the peculiar element which has changed the organ appears only as a refracting, corneous-looking substance, of no particular form or structure. In the portions of the liver less affected, this substance is seen mixed with the secreting cells

in the lobules, the cells themselves having a withered appearance, being small and almost devoid of granular contents. If the circumference of the liver has become fatty, the appearance of lobules under the microscope, with their dark opaque margins, becomes very well marked. In some cases an increase of fibrous tissue has appeared to be present, but whether this has been an essential part of the morbid change, or whether it has been due merely to a coincidence of the lardaceous degeneration with an early cirrhosis, it is difficult to say. The lardaceous liver is little prone to change; is not much affected by water, alcohol, acids, alkalies, or ordinary reagents; and may be kept for a great length of time without any odour being emitted, or other signs of decomposition manifesting themselves.

*Lardaceous spleen.*—There are three conditions of the spleen to be found in connection with the disease under consideration. Although only one, probably, can strictly bear the name lardaceous, the others have no doubt strong ties of relationship with it. The first, or true affection, is recognised by the presence of round translucent bodies, pervading the structure of the organ, occupying, in fact, the place of the ordinary splenic or Malpighian corpuscles. These bodies are about the size of millet seeds, although they vary in size according to the degree of the disease. In the most extreme cases they never occupy more than about half the bulk of the organ, the intervening pulp structure being healthy. The spleen, as a whole, is generally enlarged, though not very much so, and therefore no indication may exist externally of the disease within. In consistence the organ is hard, but not more so than is witnessed in some forms of heart disease. Even when a section is made the alteration in structure might be very readily overlooked, and no doubt often is so; but after being exposed to the air for some time, the distinction in colour between the adventitious matter and the natural splenic structure becomes very evident. These translucent bodies, as before said, are formed by the deposition of the lardaceous material in the Malpighian corpuscles, and the same product is sometimes seen surrounding the smaller arteries which pass to the latter, as well as affecting some of the fibrous trabeculæ. The material itself, both to the naked eye and to the micro-

scope, presents the same appearance as that found in the liver.

The second form of the disease is where a peculiar translucent substance pervades the organ in all parts, giving the appearance as if a quantity of melted tallow had been poured into its cellular structure. This matter appears identical in all respects with that before mentioned; but instead of affecting the splenic corpuscles by a slow transformation, is poured out into the substance of the organ in a more ill-defined and rapid manner; whether the two, however, result from the same affection presented to us under different circumstances, or whether they are dissimilar, remains yet to be proved by more extended observation. Besides the mere outward similarity of the disease to the ordinary form, its association with tuberculosis, and with a peculiar enlargement of the lymphatic glands, sometimes found coexisting with lardaceous disease, is another reason for supposing the two are closely allied. This is seen in cases Nos. 36 and 38, which suggest the question whether this peculiar matter in the spleen, as well as the lardaceous generally, may not be closely allied to the tubercular, but assuming another form; for since we see transparent tubercle and soft strumous deposit, whose identity is generally supposed, coexisting in one organ, so is it possible that a very rapid and acute exudation of inflammatory strumous matter might assume the condition seen in the spleen, and in truth we do witness a very similar substance produced around vomicæ in phthisical lungs. One peculiarity in the case No. 38, just mentioned, is that the matter in the spleen contained a fibro-nucleated new growth; and it will be observed that, in many cases of lardaceous disease, both in the liver, kidney, and absorbent glands, this tendency to the formation of fibre was recognised.

The third form of the disease which we have to mention bears some relation to those previously spoken of; but its precise connection with them has yet to be discovered. Its tie of relationship is not only through its own similarity of composition, but through tuberculosis and the peculiar enlargement of the lymphatic glands, which are sometimes found with lardaceous viscera. The spleen in this form is seen to be pervaded by a whitish-yellow, opaque, soft material,



in distinct masses of an irregular shape, and not occupying any particular portion of the organ, or running through it in continuous lines, as in the former cases. It resembles in appearance the mixture of lardaceous material and fat, already spoken of as often seen between the lobules upon the margins of the liver. This disease has been sometimes indiscriminately called tuberculous, but the adventitious substance differs considerably from true miliary tubercle in the spleen, although it resembles very closely the soft yellow strumous material found in this organ, and with which it is possible it may be identical. The great interest of the affection is its being associated with an enlargement of the lymphatic glands, producing an obscure but fatal disorder. In vol. iii of the 1st series of the 'Guy's Hospital Reports,' under the article "Abdominal Tumours," by Dr. Bright, this physician, while describing the various forms of disease displayed by the spleen, gives the following account of one variety which appears to be identical with that to which we refer, though he styles it malignant: "There is another form of disease which appears to be of a malignant character, though it varies from the more usual form of malignant disease, and which has been particularly pointed out by Dr. Hodgkin as connected with extreme disease of the absorbent glands, more particularly those which accompany the blood-vessels. The whole of these absorbent glands become larger and firmer, without any tendency to suppurate, as in ordinary scrofulous disease, or to soften, as in cerebriform disease; and, at the same time, the spleen becomes more or less completely infiltrated through its whole substance with a white matter, with almost the appearance of suet. This matter insinuates itself into the cellular structure of the spleen, but it is no easy matter to point out what particular portion of the structure receives it. A section of the organ seems to show, from the irregular forms assumed, that it fills a cellular structure, and, in some degree, takes its shape from the cells into which it enters; having less tendency to assume the form of regular globular masses, or tubera, than other malignant disease."

How these three forms of disease in the spleen are connected we will not at present undertake to say, but that an indirect connection exists is seen by the perusal of the above cases; where the second and third forms are both seen asso-



ciated with an enlargement of the lymphatic glands and with tuberculosis, while these two latter affections are constantly combined with lardaceous disease.

*Lardaceous-kidney.*—The lardaceous disease may occur to any amount in the kidney, associated with similar disease in other organs. It may arise as a primary affection, and often, in its most marked form, is found in connection with phthisis. In some of these extreme examples there also appears to be a considerable development of fibrous tissue; and this was particularly the case in two specimens referred to in a paper on “Bright’s Disease,” in vol. viii of this work. In such cases as these, the organ, when removed from the body, might be mistaken, at first sight, for a specimen of the large white kidney of Bright, but a little more minute examination will show the difference between them. The organ is considerably harder than the Bright’s kidney, and, instead of being easily lacerable, is remarkably firm and tough; the surface is uniform and smooth, and presents no mottling or white deposit discoverable by the eye; the cortical structure is seen to be much increased in extent, and this causes the whole organ to appear as if composed of one uniform albuminous substance (except where the apices of the cones appear), giving it a semitransparent appearance and a leathery consistence. These extreme cases are, however, seldom met with, but have been found associated with diseased bone, phthisis, or occurring as the primary disease. The more common form of the disease is a less or partial degree of it, and associated with a similar affection of the liver and kidneys, and met with, like them, in connection with necrosis of bone. The translucency, hardness, and uniformity of the surface is often sufficient to identify it, if present to any extent, but if in a less degree, the aid of the microscope is required to detect the presence of the disease. If a thin slice of the affected organ be taken, this instrument will at once discover the alteration by the peculiarity of the Malpighian corpuscles; these appearing by transmitted light as round transparent bodies, having a glistening aspect, and thus producing a resemblance to the spleen when the subject of the like disease. If the capsule be torn off, the lardaceous material will be seen surrounding and enveloping the capillary vessels composing the Malpighian tufts, and also the smaller

arteries which are going to form them. If iodine be added to such a section, the Malpighian corpuscles will be seen to imbibe the colouring matter, and appear as bright-red glistening bodies, set in the ordinary structure of the kidney.

With reference to lardaceous disease affecting other organs, there is no doubt that such occurs, and, as before mentioned, the microscope brings to light this peculiar transparent or colloid material under a variety of circumstances. Whether, however, the change in tissues which produces this lesser morbid condition is identical with that which causes the total alteration of the organ, to the destruction of the patient, as in the disease under consideration, is not yet proved. We shall therefore let the question rest for the present, and be content with having spoken of the great and marked changes occurring in the three important organs already described. It might be expected, however, that something further should be said with reference to the condition of the bone in this disease, whether it be the result only of the more ordinary and simpler inflammatory processes, or whether it be an altogether peculiar state connected with or tending to the production of the lardaceous affection in the internal viscera. More observations are yet required to give a positive answer to this question; but it will be seen that, in the majority of the above cases, the disease of the bone appeared to present no great peculiarities, except in some cases the tissue was very soft and gelatinous, and that in two cases, Nos. 10 and 13, a watery discharge took place from the tibiæ, and spongy ulcers sprang from their surface which had a great resemblance to carcinoma.

*Peculiar enlargement of the lymphatic glands.*—As before alluded to, the affection of these glands under present consideration is not strictly lardaceous, but brought forward in connection with disease of this kind, because either a variety of it, or having a close relationship with it. It is a form of disease which has attracted little notice, probably on account of its comparative rarity, and also on account of the obscurity and want of striking symptoms accompanying it. It is an affection, however, which produces a lingering form of fatal cachexia, and therefore well worthy of our especial attention. The disease might with propriety have been discussed quite independently, but we have brought it forward here in con-

nection with lardaceous disease, because sometimes found with the latter, and undoubtedly having some close affinity with it. (See case No. 36.) Another tie of relationship is its association with tubercular disease, as many of the above cases will show. The most interesting and important fact, however, in the history of this particular affection is the condition of the spleen with which it is associated—that before mentioned under the third variety of disease of this organ, and alluded to in the quotation from Dr. Bright's paper. A glance at the cases above given, including those from our museum, one lately occurring at Guy's, and one copied from the '*Transactions of the Pathological Society*,' will show how striking and remarkable an agreement exists between them all, both as regards symptoms during life and the appearances after death. Their uniformity is too considerable to constitute merely a coincidence of disease between the glands and the spleen, and therefore there is, without doubt, a peculiar form of affection involving these organs, accompanied by an anemic cachexia, prostration, and death. We say a peculiar affection, for although allied to the tubercular, we believe it to be one not yet recognised under the ordinary forms of disease.

The enlargement of the glands is in most cases gradual, extending sometimes over a period of two, three, or more years, and often, from commencing in the neck in weakly children, is called scrofulous. When the mischief is thus gradual in its commencement, and affecting only part of the glandular system, no marked symptoms ensue, but as time tends to its development in the thoracic and abdominal glands, a slow prostration ensues, terminating in death. The glands often reach an enormous size, much larger than when affected with scrofula, a bunch of them often being composed of separate tumours each the size of an egg. When felt during life, in their early progress, they are recognised by their peculiar elastic feel, differing both from the early hardness or the subsequent softness of scrofulous glands, and also differing from these (as far as our experience goes) by being quite unaffected by iodine. In fatal cases they have been found not only forming large tumours in the neck and groin, but accompanying the aorta its whole length throughout the body, and thus affecting all the posterior mediastinal and lumbar glands, occasionally even



following the arch of the aorta to the anterior mediastinum. Sometimes, however, none of the external glands have been affected, and the existence of glandular enlargement has not been known until after death, as in case No. 40. When these bodies are removed it is seen that they constitute distinct tumours, easily separable from each other, there being no bond of union between them, arising from any previous inflammatory action or deposition of new material amongst them. When incised their appearance is altogether peculiar, being of a yellowish colour, and having a soft, translucent aspect. Upon squeezing or attempting to tear them, however, this softness is found to be an illusion, as they are remarkably tough, and emit no juice, but only a little serous fluid. Upon cutting off a small portion and endeavouring to separate it by needles, it is found to have almost a leathery consistence. The cut surface of the glands is quite uniform, presenting no appearance of structure to the naked eye, though sometimes they contain small masses of dead material in the centre. The first idea suggested by their general aspect is that the disease is a simple hypertrophy of the gland, but a microscopic examination shows this to be incorrect, for although some glandular secreting elements may still be found, yet the main structure is a fibro-nucleated tissue; in fact, an altogether new formation, and not to be distinguished from an ordinary fibro-plastic growth—indeed, many tumours passing by this name, which the surgeon occasionally removes from the neck, appear to be often no other than glands affected with this particular form of disease. Sometimes, besides the nucleated-fibre element, some transparent, albuminous, amorphous material has been present, particularly in those cases where lardaceous disease has elsewhere existed, and this is another reason to warrant the opinion that the two affections are closely allied. In cases Nos. 36 and 38 the disease in the glands was associated with a peculiar wax-like substance in the spleen, and composed of a very similar material, being made up of a lardaceous matter and a new fibrous tissue. Its connection with marked lardaceous liver is seen in the former of these cases; with tuberculosis in cases Nos. 38 and 40, &c. The most remarkable feature, however, in the history of the disease, is that before alluded to, its close intimacy with that peculiar affection of the spleen which



contains the suet-like substance, and which slowly and inevitably leads to a fatal end. The symptoms appear to be only those of anæmia, prostration, and final exhaustion.

In what measure the lymphatic glands and spleen relatively participate in producing this result we will not venture to say, although sufficient is known of the functions of both organs to be assured that disease in either will lead to severe derangement of the system, and, moreover, connected as they are with the blood-making process, that if a slow destruction of their structure goes on, so a gradual death of the patient will as assuredly ensue. It will be seen that this combination of disease is remarkably alike in all the cases we have given, and that in none was any disease of the bone present.

Want of space alone prevents us from offering a much fuller account of the cases above given, together with more extended details of the pathological observations, and some theories respecting the diseases themselves. We have been content, therefore, with briefly stating the main facts connected with a particular class of disease, including several morbid conditions whose identity is not proved, but whose close relationship appeared to warrant the adoption of the present method of narration, in order to fix attention to them. First, there are the cases of simple lardaceous disease occurring alone; then those associated with tuberculosis; then those cases connected with disease of bone; afterwards those found in connection with a peculiar affection of the glands; and, lastly, those cases where this latter condition of the glands occurred unassociated with lardaceous disease (unless the white deposit in the spleen be a modification of it), but allied to the previous classes by a close relationship.

While writing this paper I endeavoured to find the observations of Dr. Hodgkin on a peculiar enlargement of the lymphatic glands, referred to by Dr. Bright, but have only now, and on its completion, chanced to meet with them in the seventeenth volume of the '*Medico-Chirurgical Transactions.*'

I there discover that one or two of the cases extracted from our museum have already been published, and Dr. Hodgkin, points out the connection of this disease with a peculiar affection of the spleen. Had I known this earlier I should have

altered many expressions which I have used with respect to any originality of observation on my part, but otherwise I do not know that I could have done better than again to refer to these cases, which resemble so exactly those which have come under my own notice. It is only to be lamented that Dr. Hodgkin did not affix a distinct name to the disease, for by so doing I should not have experienced so long an ignorance (which I believe I share with many others) of a very remarkable class of cases, a recognition of which would have guided both myself and others to an explanation of some more recent instances coming under our notice.

# REPORT OF THE POST-MORTEM EXAMINATIONS

OF THE

## CASES OF BURN

OCCURRING DURING THE LAST ONE AND A HALF YEAR,

WITH

REFERENCE TO THE CONDITION OF THE DUODENUM.

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BY SAMUEL WILKS, M.D.

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IN the twenty-fifth volume of the 'Medico-Chirurgical Transactions' there is a paper, by Mr. Curling, setting forth the frequency of ulceration of the duodenum in cases of burn, as shown both by inspection after death and by the vomiting and purging of blood during life. The instances given by that gentleman occurred in persons of various ages, and in whom death took place at all periods between the third day and fifth week.

The examination of those dying from this cause being exceptional, our experience of their post-mortem appearances has hitherto been limited; but whenever the opportunity for inspection has been afforded, the statements of Mr. Curling have not been corroborated. In order, however, to put the matter to a further trial, and to obtain a greater number of facts with reference to the subject, we determined at the beginning of last year to examine all persons dying of burns, and thus now we are enabled to offer twelve cases for perusal in which post-mortem inspections took place.

During the year 1855, and the first half of the present year, there have been 37 fatal cases of burns and scalds; of

these, 12 were fatal within a few hours, 5 before the second day, and 6 before the third day. The subjects of these had never recovered from the shock of the accident, and therefore no post-mortem examination was performed, as it was considered such would have been altogether fruitless. The remaining 14 cases died after longer intervals, and of these 2 only were not examined, one, a boy, æt. 3, who died twelve days, and another, a boy, æt. 2, who died eight days after the receipt of the injury. In neither were there any gastro-intestinal symptoms during life. The remaining 12 cases were as follows:

CASE 1.—Jessie A—, æt. 3. From clothes catching fire, burned her face, neck, and arms. She lived sixteen days. Bowels always regular and natural. *Post-mortem*: Brain not examined; lungs purpuric on surface; stomach healthy; *duodenum* quite healthy; slight diphtheritic condition of colon; liver and spleen purpuric on surface; kidneys contained fibrinous deposits in cortical substance.

CASE 2.—Jeremiah H—, æt. 10. Clothes caught fire, and large part of body and limbs burned. Lived nineteen days, in the early part of which period he had vomiting and purging, but passed no blood; towards the close the bowels were confined. *Post-mortem*: Body only partially examined, as no permission was given; *duodenum* quite healthy; stomach and ileo-cæcal portion of intestine healthy.

CASE 3.—Alfred C—, æt. 6. Clothes caught fire, and he burned the left side of body and leg. Lived thirty days. No gastro-intestinal symptoms. *Post-mortem*: Lungs in state of lobular pneumonia; *duodenum* healthy, as well as stomach and whole of intestine; kidneys filled with fibrinous deposits.

CASE 4.—Mary F—, æt. 6. Clothes caught fire, and upper part of body much burned. Lived eight weeks. *Post-mortem*: Body much wasted, and covered with purpuric spots; lungs, lobular pneumonia, softening; *duodenum* healthy; remainder of intestines and stomach healthy; liver fatty.

CASE 5.—Eliza H—, æt. 9. Clothes caught fire, burning upper part of body. Died of tetanus, in nine days. *Post-mortem*: Brain and spinal cord healthy; *duodenum* quite healthy, also stomach, intestines, and other organs.

CASE 6.—William H—, æt. 5. From clothes catching fire, upper part of body and face much burned. Lived nineteen days. *Post-mortem*: Lungs hepatized; *duodenum* healthy, as well as remainder of stomach, intestines, and other organs.

CASE 7.—William R—, æt. 47. Stepped into a vat of boiling tallow, and excoriated both his legs. Lived thirty-one days. *Post-mortem*: Lungs hepatized; liver, early cirrhosis; *duodenum* quite healthy, as well as stomach and remainder of intestines.

CASE 8.—George H—, æt. 15. Extensive scald of arm and leg by hot tar. Whole of integument of leg sloughed, laying bare the tendons and nerves. The limb in consequence amputated. Died thirty-three days after the receipt of the injury from pyæmia. *Post-mortem*: Lungs sloughing, lobular pleuro-pneumonia; spleen suppurating; *duodenum* healthy, and rest of alimentary canal.



CASE 9.—William M—, æt. 2. Scald of face, neck, and chest, by hot water. Lived nine days. *Post-mortem*: Brain not examined; *duodenum* healthy, as well as stomach, remaining part of intestines, and all other organs.

CASE 10.—Samuel M—, æt. 1 year. Burn on face, neck, and chest. Lived thirteen days. *Post-mortem*: Brain not examined; *duodenum* healthy, also stomach, intestines, and all other organs.

CASE 11.—Ellen F—, æt. 15. Scald on chest, abdomen, and thighs. Lived nearly seven weeks. *Post-mortem*: Lobular pneumonia; *duodenum* and remainder of intestines quite healthy; stomach, opposite the attachment of the omentum there existed an ulcer or hemorrhagic erosion nearly two inches in length, the mucous membrane was destroyed, and upon the tissue beneath was a small quantity of adherent coagulated black blood. (Similar ulcers are not unfrequently met with in a variety of diseases, where the powers of the system are very low.)

CASE 12.—Joseph C—, æt. 3. Burn on face and chest. Lived twenty-one days. *Post-mortem*: There was no permission to examine the body; but the *duodenum* was extracted, and found perfectly healthy.

It will be seen that in no case was there any disease discoverable in the duodenum after death, and that as regards the remaining part of the alimentary canal, in one case only was a slight diphtheritic condition of the colon found, and in another an erosion of the stomach. The latter form of ulcer is far from uncommon, being frequently met with in those dying from heart-disease and other causes. In none of the cases, moreover, were there any symptoms denoting severe lesion of the intestinal canal during life, and the same fact is true of all the other fatal cases which were not examined, as well as all those which recovered during the above-named period. The question naturally arises, how can the present facts be reconciled with those of Mr. Curling? This is a question, however, which we cannot at present attempt to solve. Generally, when writers differ as to facts, they doubt the accuracy of each other's observations, but such an explanation will not suffice in the present case, for it is as equally impossible for a healthy surface to be mistaken for an ulcerated one as it is for an ulcer to appear as a smooth healthy surface. On inquiry of the sister of the ward into which these cases are admitted, she states that she does not remember to have witnessed an instance of evacuation of blood from the stomach or bowels for several years, but that she can call to mind several cases where this occurred many years ago; and on mentioning this circumstance to my colleague, Dr. Habershon, he suggests whether the practice of giving children ardent spirits imme-

diately after the receipt of a burn may have been the cause of the lesion : that such was the usual habit of parents formerly, but one now generally discontinued, is also the experience of the sister of the ward. Whether this is the explanation or not we will not attempt to say, but this is clear, that the subject is still open for much more extended observations.<sup>1</sup>

<sup>1</sup> During the last session of the Pathological Society, an example of ulcer of the duodenum after burn was exhibited by Dr. Gibbon, of the London Hospital, from which it would appear that the fact does still occur at that institution. It would, however, be important to know whether this is the only case of the kind which occurred during the year, for if so, its tendency would rather be opposed to the proof of the rule in favour of the ordinary occurrence of the lesion, seeing that it would be but a solitary one among a number of cases of burn, which we believe amount to more than fifty yearly in the London Hospital.

# BRIEF REPORT OF THE POST-MORTEM EXAMINATIONS

OF THE

## CASES OF FEVER

WHICH HAVE DIED IN THE HOSPITAL DURING THE LAST TWO YEARS.

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By SAMUEL WILKS, M.D.

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THE last volume of these Reports contained an account of all the cases of fever up to the autumn of 1854. It would have been valuable, had space allowed, to have continued the report up to the present time, since the amount of fever in the hospital has been very considerable, and the field of experience proportionally large. We may state briefly that as during the last few years the greater number of cases have been of the typhoid type (only the exceptional ones being of the typhus), so during the last few months, especially in the winter, the opposite has been the fact, the prevailing form having been that of typhus. This also has been the rule throughout the metropolis. With reference to the question of specific differences between typhus and typhoid which was entered upon in the last volume, we may state that the greater number of cases being of the typhus kind, with the presence of a mulberry rash, the distinction has been well marked and the diagnosis easy; but in some of the milder forms the symptoms were considered to be not sufficiently distinctive to warrant their separation into species. Whatever difficulties, however, may have existed in the attempt to dis-sever the resemblances which may have been present during life, no such difficulties were found after death; for then a clear distinction was apparent between the two forms. In one

class of cases no disease whatever was found in the intestine, and in the other a very remarkable condition and one altogether characteristic. There has been no intermediate state or point of transition between them, but a healthy condition on the one hand and a marked disease on the other. In most of the cases the symptoms of each class were distinct, and led to the knowledge of the appearances found after death, thus tending, as we believe, to make a clear separation between them. Leaving, however, for the present, the symptoms which existed during life, and regarding merely the appearances found after death, we cannot but conclude that we are looking upon diseases of altogether different kind.

It will be observed that, with the exception of one or two cases of each class being removed from their proper period of occurrence, all the cases entered the hospital in the order detailed below, and thus it will be seen how remarkably all the typhoid cases came together during the half-years 1854-55, and the typhus more lately during 1855-56.

In the last Report, already alluded to, it is stated that no very characteristic elements could be found in the typhoid deposits of the intestine. In three of the present cases, however, where the disease was not quite advanced to its height, and especially in Case 3, where death took place at the onset of the fever, some remarkable microscopical bodies were found. These consisted of rounded cells, containing numerous nuclei, which amounted in some to ten in number. They were found equally in the deposit of Peyer's patches and mesenteric glands. Although on being shown such cells it might be imprudent to declare that they were characteristic of typhoid fever, yet at the same time these bodies in the three cases were so exactly similar to one another, and differed in so many respects from the exudation cells found in other morbid tissues, that in all probability they are peculiar to and belong to this disease. In the spleen of these cases, also, very similar cells were found. They were considered at the time to be, probably, the blood-corpuscle-holding cells of Kölliker, but these being so seldom seen in ordinary cases of disease, it is probable they too had some connection with the typhoid state. (See Plate V, fig. 6.)



## CASES OF TYPHOID FEVER.

CASE 1.—Eliza L—, æt. 20. Admitted on October 9th, 1854, and died October 15th. Ill two weeks, and kept her bed one week. Extremely ill; rose rash, diarrhœa, &c. Ordered wine and ammonia. *Post-mortem*: No maculæ on body; lungs extremely congested; Peyer's patches much enlarged by deposition of a soft brown deposit within the glands; the enlargement commenced at one foot and a half from termination of ileum, and proceeded downwards in an increased degree, until near the valve, where the material was being thrown off, and ulceration of the mucous membrane had commenced; also the solitary glands in the cæcum were occupied by the same deposit; other organs soft, but otherwise healthy.

CASE 2.—Abraham B—, æt. 21. Admitted October 19th, 1854, and died November 30th. On admission, ill twelve days, and the case ran the ordinary course of typhoid fever. On November 2d, he began to improve, and slowly convalesced. On the 29th, he walked about the ward, and appeared to be rapidly recovering his health; but in the evening of this day, after going to bed, he suddenly expired. *Post-mortem*: No maculæ on body; brain and lungs healthy; heart empty and flabby, but no disease discoverable to the eye or microscope; the ileum contained a large number of healing ulcers, in the situation of Peyer's and solitary glands; they were small, with puckered edges, and most of them covered with a new membrane.

CASE 3.—James L—, æt. 27. Admitted December 27th, 1854, and died December 28th. He was an intemperate man, leading a very irregular life; but no history was known of his present illness, except that he had been complaining some days; also, that two days before admission, he was out in the wet, and returned to his lodging very ill, with great difficulty of breathing, and was afterwards brought to the hospital. He there only lived a few hours. *Post-mortem*: Brain healthy; lungs much congested; the last two feet of ileum most extensively diseased; the solitary and Peyer's glands being swollen to an enormous extent, by the presence of a soft material, brown and flocculent in the centre, and of a lighter colour at the circumference; the three Peyer's patches at the termination of the ileum were so swollen by this deposit, that they completely filled the calibre of the intestine; a few solitary glands in the cæcum were raised into buttons by the same deposit; the mesenteric glands in proximity to the lower portion of ileum were very much enlarged and soft. A microscopic examination of the deposits in Peyer's and mesenteric glands displayed rounded cells, containing from three to ten nuclei.

It will be seen that this man died at the onset of fever from exposure to cold, and thus a valuable opportunity was afforded for witnessing the condition of the ileum at a stage prior to that in which it is generally examined. In most cases ulceration has occurred at the time of death, whereas in the present instance the material formed in the glandular structures had not yet sloughed out, but existed as large masses of soft granular and cellular substance, almost closing the intestine. The mesenteric glands were also seen to be affected in a

similar way. It was also a good occasion to test the material by the microscope.

CASE 4.—Mary W—, æt. 29. Admitted January 15th, 1855, and died January 23d. Been ailing two weeks, and had kept her bed one week. Gradually sank. *Post-mortem*: Brain healthy; lungs much congested; ileum and cæcum extensively diseased; large raised deposits in Peyer's glands about one foot and a half from the valve, these were sloughing in the centre; lower down ulceration had occurred, and the last six inches of the ileum had its surface raised by this soft brown flocculent deposit; in the cæcum there was a similar deposition, and ulceration in solitary glands; mesenteric glands much enlarged, red, and soft.

CASE 5.—Sarah B—, æt. 16. Admitted February 14th, 1855, and died March 5th. This girl had always been delicate, with a cough, and had been very ill for three weeks, with symptoms of fever. She had severe bronchitis, diarrhœa, bed-sores, &c. She gradually sank. *Post-mortem*: Lungs much congested, and posterior parts in early state of hepatization; ileum, for its last five feet, presented an ulcerated surface; the ulcers occupied solely the patches of Peyer and solitary glands; the usual brown deposit had nearly disappeared, and thus they presented merely an ordinary appearance with raised swollen edges; near the valve they were slightly contracting, as if the healing process had commenced; mesenteric glands opposite ileum enlarged and soft. (Probably, in this case, the height of the fever was passed, and death occurred from broncho-pneumonia and the bed-sores.)

CASE 6.—Joseph K—, æt. 17. Admitted June 20th, 1855, and died July 6th. Had been suffering from cough with expectoration for two months, and been very ill a few days before admission. Then he was too ill to give any account of himself; but he had a rose rash, diarrhœa, and bloody expectoration. *Post-mortem*: No maculæ apparent within the inked circles marked around them during life; lungs in state of red hepatization; Peyer's and solitary glands in all degrees of disease; about one foot and a half from termination of ileum, the enlargement of those parts began by the presence of a deposit within them, and continued downwards until they became ulcerated near the valve; the solitary glands also in large intestine felt like peas in the mucous membrane from the presence of this deposit.

CASE 7.—William K—, æt. 16. Admitted October 10th, 1855, and died October 19th. The boy was in a half-conscious state, and was said to have been ill a fortnight. He had all the usual symptoms of typhoid fever. The *post-mortem* showed acute peritonitis from perforation of ileum four inches from its junction with the cæcum; the former presented the usual appearance of typhoid affections of the glands, the disease commencing about two feet from the termination; at the lower part, this was sloughing out, leaving an ulcerated surface, in the middle of one of which was a pin-hole perforation; mesenteric glands enlarged, red, and soft.

CASE 8.—James C—, æt. 26. Admitted November 20th, 1855, and died November 27th. Too ill to give any very clear account of himself, but stated that he had been ailing three weeks, and had desisted from work one week. Well-marked typhoid fever. Suddenly grew worse and died. *Post-mortem*: Showed no general peritonitis, but a few coils of intestine in the right iliac region were adherent by a slight and very recent exudation; the bowel within presented the usual appearance, the highest glandular patch filled with deposit being three feet from the cæcum, the disease being more advanced towards the latter part; in an ulcer near the valve, the

peritoneal coat was translucent, and though no actual perforation was visible, no doubt a transudation had occurred through it, leading to the peritonitis and rapid death; mesenteric glands very much enlarged.

CASE 9.—John H—, æt. 27. Admitted February 20th, 1856, and died February 29th. He stated that eight days before admission he got very wet, and was afterwards seized with shivering, &c. He fell into a febrile state, and so continued until first seen, when he was found very ill, a few rose spots making their appearance on abdomen, and diarrhœa. He also had a severe cough, which got much worse, with great rapidity of respiration. *Post-mortem*: Lungs much congested; ileum had Peyer's patches much enlarged by adventitious deposit, being of an oval shape, and considerably raised above the surrounding surface; in some the mucous membrane was ulcerated, and from them protruded a brown granular material; the last one near the valve had sloughed out, leaving an irregular excavated surface; in cæcum and commencement of ascending colon there were about a dozen solitary glands affected in a similar way, being filled with a peculiar material, and feeling like peas beneath the membrane. Mesenteric glands much enlarged.

CASE 10.—Charles S—, æt. 38. Admitted November 21st, 1855, and died April 2d, 1856. He was admitted with typhoid fever, had rose spots on his body, and other symptoms. The disease was severe, and his recovery was very protracted. When about to leave the hospital he was seized with typhus, accompanied by a well-marked mulberry rash, and he quickly died. *Post-mortem*: There was no recent disease in the intestines, but in the lower part of the ileum, occupying the position of Peyer's glands, were several cicatrices of ulcers; these were small, smooth, puckered patches. (The date of the inspection was nineteen weeks after the first symptoms of the typhoid fever.)

CASE 11.—John J—, æt. 27. Admitted June 27th, 1856, and died July 11th. He was an intemperate man, and said to have been ill twelve days on admission; he then was scarcely able to articulate, in a high state of fever, body covered with rose maculæ, and diarrhœa. Stimulants at once ordered. He gradually sank lower until July 6th, his cough becoming very severe. After this time the fever abated, and it was thought that he would recover; the breathing, however, became more difficult, and attended by a catch in the side; the mucus collected in the bronchial tubes, and thus he died. *Post-mortem*: Body that of a strong-looking young man; no eruption visible; pleuro-pneumonia on the left side, with great congestion of the lungs; ileum, the lower Peyer's patches were completely excavated by ulceration, particularly those nearest to the valve; there was no appearance of adventitious deposit about them, nor of sloughing, but the edges were slightly raised, puckered, and contracting; the floor was composed of the muscular coat, and upon it were some soft granulations. (The appearance of healing presented by these ulcers corresponded to the history of death having taken place five or six days after the height of the fever had passed.)

## CASES OF TYPHUS FEVER.

CASE 12.—William F—, æt. 28. Admitted January 2d, 1856, and died January 11th. On the 28th of December he was seized with severe headache and fever, and continued growing worse until admission; at that time he was covered with a mulberry rash; he had a cough and bloody expectoration, and on the 4th the rash had become



petechial. He daily grew worse; wine, ammonia, and brandy were administered; and he died on the fourteenth day. *Post-mortem*: Petechiæ on body; brain, a strumous tubercle existed in the cerebellum; lungs much congested; ileum and intestinal canal quite healthy; other organs structurally healthy.

CASE 13.—John G—, æt. 24. Admitted February 20th, 1856, and died February 24th. He was too ill to give any account of himself, but he had led a dissipated life, and had lately got wet through. On admission, was in a high state of fever, delirious, and covered all over with a mulberry rash; he rapidly got worse, having some convulsive movements before he died. *Post-mortem*: One or two spots on legs; the brain and intestines were alone examined, and these were found quite healthy.

CASE 14.—Alfred B—, æt. 26. Admitted March 17th, 1856, and died March 23d. On the 9th he took a long walk, and sat upon the damp ground; on the following day he felt unwell, being seized with headache, feverish symptoms, &c., and was obliged to give up his work; on the 17th he came to the hospital, covered all over with a mulberry rash, and some difficulty of breathing. He gradually grew worse; the rash, which was universal, became petechial, and the breathing became more rapid. A large amount of stimulants was administered, but he died on the same day fortnight on which he over-fatigued himself and exposed himself to cold. *Post-mortem*: Numerous petechiæ on body, and particularly on legs; no structural disease in any organ; the ileum and other parts of intestine quite healthy.

CASE 15.—Henry M—, æt. 35. Admitted May 14th, 1856, and died May 22d. He was taken ill on May 5th, and in three days took to his bed; on admission he was excessively ill, could with difficulty speak or protrude his tongue, tremor, and skin covered with a mulberry rash. Stimulants were given, but he continued to decline until the 19th, when all the febrile symptoms were less and the eruption was fast fading; his mind also became clear. The extreme debility, however, continued, and the respiration much accelerated, and he died two days afterwards. *Post-mortem*: Lungs extremely congested; a small extravasation of blood beneath capsule of spleen; intestines quite healthy.

CASE 16.—Hannah E—, æt. 30. Admitted on May 24th, 1856, and died on the 30th. She was received into the accident ward, on account of injuries she had inflicted on her neck during the delirium of fever; these were, however, slight. She was delirious, very low, and covered with a mulberry rash. She gradually sank, in spite of stimulants, the respiration at last becoming much accelerated. *Post-mortem*: The posterior part of lungs in a state of red hepatization; intestines quite healthy.

CASE 17.—Elizabeth R—, æt. 59. She was a nurse in the hospital, but had been attendant on no case of fever. She was taken ill on the 16th of May, 1856, and on the 21st, when she became a patient, was in a high state of fever, with a well-marked mulberry rash. She daily got lower, in spite of nourishment and stimulants, and died on the 1st of June. She was said to have had diarrhœa on one or two occasions during her illness. *Post-mortem*: No inspection was allowed, but by a small incision the ileo-cæcal portion of intestine was extracted, and found perfectly healthy.

CASE 18.—James O—, æt. 47. Admitted June 18th, 1856, and died June 20th. Was the subject of heart disease, albuminous urine, and slight dropsy; he was taken ill a week before admission, and rapidly sank into an extreme state of fever. On admission, universal mulberry rash, dark tongue, delirium, &c. *Post-mortem*: Lungs extremely congested; heart much affected by enlargement, diseased valves, and adherent pericardium; kidneys granular; intestines healthy.



# CASES OF PARAPLEGIA.

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By WILLIAM GULL, M.D.

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## PARAPLEGIA FROM TUMOURS COMPRESSING THE CORD.

TUMOURS growing in the cord, or from its membranes, are among the more rare causes of paraplegia. With the exception of scrofulous deposits, these formations are most frequently seated in the loose tissue under the visceral layer of the arachnoid, or grow from the inner surface of the dura mater. They have generally been regarded as malignant, but their microscopical characters, their indisposition to invade or infiltrate surrounding parts, and their non-occurrence simultaneously in other organs, refer them to a simpler class of tumours, the *fibro-nuclear* or *fibro-cellular*. The cord and its membranes appear to be extremely rarely affected primarily by cancerous growths. When malignant disease attacks these parts it is generally by secondary diffusion, or by extension from the bones or other structures adjacent.

In paraplegia from compression of the cord by tumours, pain is, with but rare exceptions, a prominent and characteristic symptom. Some writers have expressed an opinion that it is present only when the membranes or surrounding structures are implicated, and not when the disease is strictly limited to the cord itself. This does not appear to be a well-founded distinction. In a case recorded, by Mr. Shaw, in the 'Transactions of the Pathological Society' (1848-49), paraplegia was produced by two scrofulous tubercles occupying the *interior* and lower part of the spinal marrow, and *invested all round by a thin layer of medullary matter*; yet the patient complained so much of pain in the lumbar region, that it was thought her symptoms might arise from caries of the vertebræ. After

death the membranes and bones were found healthy. The character of the pain appears to be very variable. In one case, quoted by Abercrombie, the first symptom was neuralgic pain in the arm, which diminished as paralysis came on. In another the patient had sciatic pain extending to the toes. Mostly the pain is referred to the back, and more or less correctly indicates the seat of the disease, from which it radiates in the direction of the nerves whose roots are invaded. Where there is no actual pain there may yet be other modifications of sensation, as coldness, or heat, or sudden alternations of these, and many other varieties of impaired feeling.

Next to pain is the frequency of muscular contractions in the affected limbs, followed as the case progresses by flexion and rigidity, and attended by a great susceptibility to the excitomotor stimulus. These phenomena are most apparent where the cord is merely stretched or compressed, and where no other change has occurred in it beyond atrophy, the communication with the brain being at the same time not entirely destroyed. If there be inflammatory softening of the substance of the cord, then these more characteristic symptoms may be absent, as they are also in compression of the cord from fracture when its structure is bruised and softened. Spasmodic contractions of the muscles of the extremities occur whether the fibres of the cord are compressed by tumours on its surface or stretched by tubercle deposited within it. At one stage of a case there may be rigid extension, which may be gradually followed as the case progresses by as rigid flexion, though the muscles at the same time may become atrophied and flaccid.

The vagueness of the early symptoms, in these as in other cases of paraplegia, deserves especial consideration in a practical point of view. In the first case here recorded, the early symptoms, cough and slight dyspnœa, and some pain in the back and shoulders, were referred to tubercular disease of the lungs. In the second, the spasmodic action of the limbs was so great that for a time the case was regarded as one of hysteria. In fine, the symptoms of neuralgia, hysteria, lumbago, rheumatism, phthisis, colic, renal calculus, pleuritic and hepatic affections, may rise like so many phantoms, to delude us at the outset of most paraplegic affections, and the errors they are apt to lead to can be

avoided only by a rigid inquiry. Of the diseases of the nervous system in general, and of paraplegia in particular, it may be said that there is no symptom or single group of symptoms which, taken alone, can serve as a secure basis of diagnosis; the whole particulars included in the clinical history and present state of the patient must be viewed in their relation to each other and to *time*, before we can discern the truth they indicate.

**CASE I. —PARAPLEGIA.**—*Tumour growing from the inner surface of the dura mater of the cord. Early symptoms, simulating Incipient Phthisis, subsequently Rheumatism.*

Francis H—, æt. 30; a married man, of temperate habits; by trade a baker; admitted into Guy's Hospital, January 30th, 1850, with symptoms supposed to be due to incipient phthisis. He had had cough and shortness of breath for two months; lost strength and flesh rapidly, and had frequent perspirations. The cough was accompanied by pain in the upper part of the back and in the right shoulder, but the complaint he made of it was not such as particularly to arrest attention. The heart's action was normal; respiration 24. He was ordered to take cod-liver-oil. There was nothing specially noticed in his symptoms until the 4th of February, when he suddenly found himself unable to empty his bladder; he was relieved by the catheter, and was not for some time again troubled in that way. A week subsequently the report says, "he is improving under the use of the oil." On the 20th, the pain in the right shoulder became much more severe, and he complained of feeling very languid, though up to this date he was able to walk about a good part of each day. He had occasional rigors and profuse sweatings. The increase of his symptoms at this time was attributed to his having taken cold. The next day the knees were painful, and the legs weak, he could not support himself, though he had the power of moving freely in bed. The character of the affection of the joints was such as to induce the belief that he was now labouring under rheumatism, and he was treated accordingly, apparently with good effect. At this time the inability to pass water returned, and decided symptoms of paraplegia came on, with impairment of sensation as high as a line round the chest, corresponding to the third rib; the boundary of the anæsthesia was not, however, sharp and defined. The arms were slightly enfeebled. The paralysis of motion in the lower extremities and sphincters became complete, but he retained the power of distinguishing the seat and direction of superficial impressions on the skin, though no amount of pinching or pricking gave rise to pain. The spinal column was straight, and no tender spot could be discovered on percussion, or by the application of a hot sponge. On the 9th of March, the urine was ammoniacal and contained blood. The rigors continued, and he had constant and profuse sweatings. A large slough formed over the sacrum. The pulse became frequent and feeble. He died rather suddenly on the 22d, about four months from the date of his first symptoms.

*Sectio cadaveris.*—Cord softened to the extent of three



quarters of an inch opposite the first dorsal vertebra. A careful examination of a transverse section showed the softening to be general, the anterior parts not being apparently more affected than the posterior. The softening seemed to be due to diminished nutrition from pressure, no traces of inflammatory exudation being detected by the microscope. The arachnoid was healthy. Attached to the inner and anterior surface of the dura mater, opposite the softened portion of the cord, was a small tumour of the size of a hazel nut. It was vascular, and consisted of nucleated cells and free nuclei in a slimy albuminous blastema. An irregular cyst existed in the centre of the mass. The bones and ligaments of the cord were healthy. The bases of both lungs were consolidated by pneumonia of recent date; there was no trace of chronic disease. Liver soft; tissue injected. Urethra healthy. Four false passages into the bladder. The pelves of both kidneys and the bladder full of a bloody purulent secretion. The mucous membrane dark and sloughy. The secreting portion of the kidneys variegated with spots, and irregular lines of pus in the suppurating tubules.

*Remarks.*—An inspection of the chest movements, even in an early stage, and before the more decided symptoms of paraplegia came on, would doubtless in this case have elucidated the cause of the dyspnœa and cough. In a similar case which came under my notice, the patient was supposed to be labouring under ordinary bronchitis with dyspnœa, when I was requested to see her; but there the partially paralysed movements were at once obvious when the chest was exposed. I have to record another case, one of induration of the cord, where the early symptoms were referred to the chest, and supposed to be phthisical. The difficulty of detecting diffused miliary tubercles with the stethoscope, and the possibility of their existing without producing any perceptible dulness or marked flattening of the chest, favoured the erroneous inference of phthisis which was drawn in these cases from the cough, emaciation, and perspiration. The suspicion of such a fallacy, with a scrutiny of the thoracic movements which I have hinted at, would guard against a similar error. The cough of hysterical subjects, which is often accompanied with tenderness in the upper part of the dorsal region of the spine, may receive



some elucidation from the early symptoms in this case. That the cough and dyspnœa had here a spinal origin seems evident, although, doubtless, in the progress of the case, the impeded respiratory movements, and the consequent congestion of the lungs, had a great share in increasing the symptoms. The rigors and sweatings which characterised the middle period of the case were probably due to the secondary morbid changes in or about the urinary passages set up by the retention of urine and the injuries from catheterism, and not to stretching or compression of the structures of the cord by the tumour; at least it is noticeable that they were subsequent to the first catheterism, and in most cases they seem to be owing to secondary lesions. I shall not now venture to call the growth found in this case *malignant*, although at the time I examined it I had no doubt that such was its nature, and similar growths are so described by authors. I have reason to think, that before we pronounce so categorically on these productions, we must know more of the individual pathology of the membranes of the brain and cord. There was no trace of a repetition of the disease elsewhere, as commonly occurs in cancer. This fact, together with the frequency of such tumours in the dorsal region, where the effects of mechanical injuries are most felt, and the age of the patient, render it probable that it was, as its microscopical structure indicated, a simple growth due to some *local* cause of irritation. In a series of cases we may notice the gradations from nuclear and cellular towards fibroid, fibrous, and bony, in the character of these productions, such varieties being probably due to the rapidity and seat of the formation. In this case the structure was such as indicated rather rapid growth, and the history of the symptoms corresponded to it, ranging over a period of about *four months*. In a parallel case of tumour from the theca vertebralis producing paraplegia, recorded in the 'Transactions of the Pathological Society' (1847-48), the symptoms had a course of *five years*, and there the structure of the tumour was of a *firm consistence*;—*osseous* where it sprang from the dura mater, and at the other parts *fibrous*, with rough granular matter intermixed. With respect to treatment little is to be said. A better pathology and a more correct diagnosis will lead to better if not to more successful methods. At present there

are no cases where treatment is more loosely tentative and empirical, than in cases of paraplegia where the causes are obscure.

CASE II.—PARAPLEGIA.—*Fibro-nuclear tumour (fibro-plastic) growing from the inner surface of the dura mater of the cord, opposite the third dorsal vertebra. Bones and ligaments healthy.* (See Plate IV, fig. 2.)

Sarah A—, æt. 43, was admitted into Guy's Hospital, July, 1855, under the care of my colleague, Dr. Hughes (whose kindness I have to acknowledge in allowing me to make use of this case). She was a healthy-looking woman, of a fair complexion, rather below the middle stature, employed as a domestic servant. In January, she first felt pains in the shoulders, chest, and sides, aggravated at night, and at the time vaguely attributed to cold. She applied as an out-patient at a dispensary, and was repeatedly blistered between the shoulders without benefit. Her strength failing, she went into the country, and kept her bed for a fortnight, hoping to obtain relief by rest. Her symptoms gradually increased in intensity, and she now began to suffer from spasmodic contractions of both lower extremities, but especially of the left. After a short time the legs were permanently drawn up to the abdomen; and, according to her description, the cramps and spasms extended to the abdominal muscles. On admission the legs were flexed, with the heels to the nates, nor could they be extended without considerable force. Left to themselves after extension, they were suddenly jerked, or more slowly drawn up into their former position. No affection of sensation. General health good. Respiratory and cardiac sounds normal. Catamenia regular. No incontinence of urine; but difficulty in voiding it; the secretion normal in appearance and acid. Constipation. Tongue clean. Appetite good. Great distress, especially at night, from the spasmodic contractions of the legs and abdominal muscles. Her sex, her healthy aspect, the absence of any deformity of the spine, and the spasmodic character of the symptoms, led to the suspicion that there was spinal irritation of an hysterical or functional character, rather than organic disease. Three days after admission she had retention of urine. On the 1st of August menstruation returned normally. Her general health appeared unaffected. She still made great complaint of restless nights from the spasms in her legs, and of a burning pain between the shoulders, extending round to the abdomen. The sensibility of the lower extremities was unaffected. On the 6th she was cupped over the spine without relief. The legs were permanently drawn up to the nates. The urine dribbled slowly away. After a few days, bed-sores began to form over the sacrum, and the urine became ammoniacal and loaded with mucus and pus. On the 24th she had constant vomiting, and the tongue became dry. The spasms in the lower extremities decreased, but the great and incessant pain in the back, and the burning pain in the abdomen, prevented her getting any rest. Her strength gradually declined; very extensive sloughs formed over the sacrum and hips, exposing the bones beneath to the extent of several inches. She died extremely emaciated on the 15th of October, about nine months from the beginning of her symptoms.

*Sectio cadaveris.*—Bones and ligaments of the spine healthy. The theca vertebralis much distended with fluid. In the dorsal region it was translucent, and speckled with granular opacities on its posterior surface. Opposite the second and third dorsal vertebræ, the cord was pushed backwards, and compressed and flattened by a smooth oval tumour growing from the inner surface of the anterior layer of the dura mater. The tumour had much the appearance, and was about the size, of a child's testicle. It had not invaded the textures, nor caused any absorption of the anterior columns. These were somewhat softened, and separated by the widening of the anterior fissure, but still everywhere continuous. Above and below the seat of compression, the cord, though small, had its natural firmness and form, and the tubules of the roots of the nerves and of the columns, examined microscopically, were normal. At the part softened by pressure, the columns contained *granular matter* and *granule cells*, scattered amongst the tubules. Over the arachnoid, on the posterior surface of the cord, there were several scattered fibroid plates. The spinal fluid was greatly in excess, but became only very faintly opalescent by heat. The structure of the tumour was firm, and consisted mostly of cohering nuclei, generally oval, but in the firmer parts linear, with a small amount of intervening granular blastema, which, in parts, had become incorporated with the nuclei into an obscurely fibrous structure (Plate V, fig. 1). In one or two parts near the surface of the tumour, the texture was softer, and collections of granular matter and a cell-wall were formed around the nuclei. The whole tumour was vascular, and on compression gave out a slight quantity of clear moisture, but no opaque juice as in cancerous formations. The lungs, heart, liver, and intestines were healthy, but wasted. Kidneys not enlarged; their tunics slightly adherent; the surface granular, with a few obscure points of commencing suppuration in the tubules. The mucous membrane of the pelves and bladder injected, and covered with puriform exudation. The walls of the bladder thick. The lesion of the urinary organs was very moderate in comparison with the very extensive sloughing about the nates and trochanters. The mucous membrane of the rectum was covered with muco-purulent exudation, but otherwise healthy.



*Remarks.*—The prominent symptoms in this case, after the pain in the back and shoulders, were the painful cramps and spasmodic contractions in the lower extremities and abdomen. In a case of a similar tumour compressing the lower part of the cord, in a young woman whose case is quoted by Abercrombie from Gendrin, the patient suffered acute pain in both legs, and convulsive retraction of the toes, and the sensibility of the left foot was so exalted that the slightest touch produced a sense of laceration. It appears that the cord, when encroached upon by a tumour which lightly stretches or compresses it, reacts as a nerve does; if the disturbance of the structure be but moderate, there is spasm and neuralgia, passing on, with increase of the lesion, into paralysis and anæsthesia. Those who hold the theory that a motor function attaches to the anterior columns, and a sensitive function to the posterior columns, will find, to some extent, a confirmation of their views in this case, in which the lesion of the anterior columns was attended by an early and marked disturbance of the motor functions. The symptoms do not, however, seem to admit of so limited an explanation. In 1848 I took occasion, in some lectures then published, to show that we had no clinical facts, which, fairly looked at, could be so interpreted; but that, in disease of the cord not implicating the trunks of the nerves or their roots, the motor function generally suffers first, whether the lesion be in the anterior, the posterior, or the lateral columns. My further experience has confirmed this statement, which has of late received elucidation from the experiments of M. Brown-Sequard. We cannot therefore form any diagnosis of the seat of disease in respect of the columns of the cord in cases of paraplegia from the loss of motility preponderating over the loss of sensation, since this happens as a constant phenomenon in all affections which are limited to the cord proper. The opinion which I have already expressed about the nature of these tumours, in the remarks appended to the last case, receives confirmation from the histology of this one. It was a vascular tumour, with a nuclear, fibro-nuclear, and partly cellular stroma, not invading the tissues around it, and not repeated in any other part, and hence probably of a simple nature.



CASE III.—PARAPLEGIA.—*Fibro-plastic tumour developed under the arachnoid, on the posterior surface of the cord, opposite the seventh and eighth dorsal vertebræ. (Plate IV, fig. 1.)*

William P—, æt. 41, admitted under care of Dr. Addison, 25th of April, 1838. A moderately muscular man, of healthy family, and until the commencement of his present illness his own health has been remarkably good, though his habits have been intemperate. He never remembers to have injured his back, but his employment as a blacksmith subjects him to laborious, and often violent exertion. He attributes his present state to drinking cold water when heated five years ago, but however this may be, about that time he became generally weak, and had cough, attended with some expectoration and pain in the left side. His symptoms were not so urgent as to induce him to seek medical aid, and after three months' rest he returned to his work. This soon brought on pain in the back and left loin, with some difficulty in walking. For six months these were his only symptoms. He afterwards began to have a feeling of coldness in the legs, and occasional loss of sensation. The pain in the lumbar region much increased, and prevented his bending the spine in stooping. In June, 1837, in addition to an aggravation of all his former symptoms, there was partial loss of power in the left leg, the right, however, still preserving its integrity until the following Christmas, when it also became similarly affected, and both were frequently subject to spasmodic jerkings and twitchings. The sphincters became weak, and day by day, up to the time of his admission into the hospital, he noticed an increase in the paralysis, and of the involuntary contractions of the legs, any attempt at voluntary motion bringing on the spasms in an aggravated form. On admission, the legs were completely paralysed, and also the rectum and bladder, and there was impairment of sensation as high as the crista ili of either side. The loss of sensation was less in the left leg than in the right, where, excepting at the posterior part of the tibia, it was complete. The least contact of the soles of the feet with the floor caused a spasmodic tremulous agitation of the legs, which, even when not thus excited, were often thrown about by spontaneous spasms of the muscles. Nothing abnormal in the form or direction of the spinal column. Respiration hurried. Heart's force augmented. No abnormal sounds. Pulse 80. Tenderness on pressure in either hypochondriac region. A fortnight after admission, pneumonia of both lungs set in, soon followed by depression, and he rapidly sank.

*Sectio cadaveris.*—A tall, wasted body. The theca vertebralis opposite the seventh and eighth dorsal vertebræ distended for rather more than two inches, and of a venous colour, from many tortuous vessels distributed upon it. When laid open, the two arachnoid surfaces were adherent at this part, but elsewhere the membranes were healthy. On the posterior surface of the cord, and covered by the arachnoid, was a large elongated vascular tumour, slightly translucent. This growth had been developed in the pia mater. It consisted of a soft yellowish substance, very readily broken up, with numerous

flattened cellular spaces interspersed through it. The medulla beneath was entire, but flattened by compression. The growth had not destroyed or invaded the membranous coverings. Under the microscope (Plate V, fig. 2) it was seen to consist, in some parts, of fine wavy fibrous tissue, imbedding elongated nuclei; in others, the nuclei were round or oval, and only loosely held together by granular blastema. The right pleura was partially coated with a layer of recent fibrin; there was a similar exudation, but to a less extent, on the left pleura. The upper lobe of the left lung was solidified, and of a reddish-gray colour, from recent pneumonia. On pressure, the pulmonary tissue gave out a grayish puriform fluid. The lower lobe on the same side, and the right lung, more or less extensively throughout, were affected with pneumonia in an early stage. Liver large, structure granular from commencing cirrhosis. Spleen large. Kidneys large, tissue injected.

*Remarks.*—The almost complete identity of symptoms in this case with those of the preceding, though in one the tumour was on the posterior, and in the other on the anterior part of the cord, may be noticed as bearing upon the remarks before made on the functions of the columns.

The fatal affections of the chest, so common in paraplegia, have probably a pathological meaning of much wider extent than our present pathology seems to recognise. I allude to the influence of the spinal cord on the pulmonary plexus, and to the probable origin of pneumonia from paralysis, or a similar state of its centres and intercommunicating cords. If not in the present number of the Reports, I hope in a future one to illustrate this subject, by cases of pneumonia having particular characters and apparently caused by disease, as aneurism or tumour, invading the trunks of the pneumogastries and the pulmonary plexus.

#### INFLAMMATION OF THE SPINAL MEMBRANES.

The more rare form of inflammation of the spinal membranes is where the dura mater is principally affected. This may arise from injuries to the column itself, or from cold, or phlebitis, or other causes. A remarkable instance is recorded by Mr. Simon in the 'Pathological Transactions' for 1855. A girl, æt. 18, had a fall, but soon recovered from its effects

and walked home, a distance of three or four miles. After eleven days pain in the back came on, with vague symptoms of pain and tenderness over the body not altogether unlike hysteria. The movements of the trunk in bed were difficult. This was soon followed by numbness and twitching in the extremities, and after a few hours by paraplegia, complete in the legs and to a marked degree in the arms. The patient died on the fourth day from the beginning of the symptoms. Suppuration had taken place outside the dura mater throughout the whole length of the spine, and, as in the case below, (Case iv) there was a burrowing of pus outwards along the course of the nerve trunks towards the mediastinum and among the muscles. The inflammation in this case was limited to the outer surface of the dura mater, and appeared to have been set up by fracture of the body of the last cervical vertebra without displacement. The record of this case is accompanied by a still more remarkable one by Dr. Bristowe, where the suppuration was not limited to the outside of the theca vertebralis and to the formation of extensive burrowing abscesses in the course of the nerve trunks, but the cavity of the arachnoid was also full of pus. In this instance the history was obscure, and the post-mortem examination threw no light upon the exciting cause. In the following case a similar state of things existed, set up, as clearly as could be indicated, by exposure to fatigue, wet, and cold. This may appear but a vague causation for so formidable a malady, but the evidence of other inflammatory affections confirms its truth. Almost every day's experience affords illustrations of pleurisy and pericarditis referable only to such a source, though pathology is at present at fault in unveiling the steps which lead to the results. To call them "*idiopathic*" is to satisfy ourselves with a term without meaning, and to call them "*rheumatic*" is to impose upon ourselves the fallacy of the "*ignotum per ignotum*." I anticipate that we may hereafter be able to trace more of these acute affections to chronic diseases, the local influence of which is at present overlooked. Such an opinion is confirmed by a survey of already recorded cases. For instance, the first case which Abercrombie himself gives in illustration of "meningitis of the cord," and which he speaks of as "an example of idiopathic acute inflammation," was almost cer-



tainly set up by phlebitis of the cervical veins from chronic disease of the ear. Local phlebitis as a source of acute disease has not, except in the instances of the liver and brain, received so much attention of the profession as its importance deserves. In some cases of paraplegia associated with gonorrhœa, lately laid, by me, before the Medical and Chirurgical Society, this was shown to be their origin; phlebitis of the vesical and pelvic veins extending to the veins of the spine, and setting up inflammation of the membranes of the cord.

**CASE IV.—PARAPLEGIA.**—*Acute inflammation of the spinal membranes. Softening of the substance of the cord.*

Charles H—, æt. 23, fair complexion, light hair and eyes; was quite well until Monday, April 21st, 1851. He spent the evening, and part of the night of that day, at Stepney fair, walking about for many hours in the wet and cold, and afterwards sleeping in his wet clothes. He affirms he did not get drunk nor receive any injury. The following day he was very unwell, with pain in the back, extending round the lower ribs, and with aching of all the limbs. The third day he was unable to leave his bed from weakness of the lower extremities, and numbness extending round the abdomen as high as a line an inch above the crista ilii. On the fourth day he began to lose power over the bladder, and the urine was afterwards drawn off by the catheter. He was admitted into the hospital on the ninth day from the commencement of his symptoms. There was then complete paralysis both of motion and sensation of the lower extremities, with paralysis of the sphincter ani and entire loss of power over the bladder. The legs were cool, and the skin mottled as from cold. He lay supine, with the legs extended. Breathing rather short and interrupted. Pulse 76. Tongue furred, white. On the tenth day (May 1st) towards evening, the skin became very hot, and the pulse rose to 132. Respiration 30. Abdomen tympanitic. No excito-motor movements could be produced in the legs, which lay extended and motionless; the muscles flaccid. Eleventh day (May 2d): Hands cold. Pulse feeble, 132. Tongue dry and brown. Urine drawn off by the catheter, acid, sp. gr. 1024. Vomits green bilious fluid. Evacuations involuntary. Slight oppression of the brain. Respiration by the superior ribs. No abdominal movement. Twelfth day: Insensible. Respiration gasping. Pulse very rapid and scarcely perceptible. Slight convulsive movements of the hands. Pupils active. Throughout the progress of the case, after admission, no twitchings or spasmodic movements in the legs, nor any to be excited by pinching or pricking the skin, nor by the application of heat. Died early on the thirteenth day.

*Post-mortem examination by Dr. HABERSHON.*—Head not examined. On making an incision into the lumbar muscles pus was found upon the laminæ of the vertebræ. The spinal canal, external to the membranes, was filled with pus from the first dorsal vertebra to the third or fourth lumbar. There was



a thick uniform coating of pus over the whole of the dura mater, but principally on the posterior aspect, except one patch about the first and second lumbar vertebræ. In the dorsal region pus surrounded the nerves as they left the canal. The dura mater was much thickened, and of a dull white colour, except in some parts, which were beautifully injected. At the commencement of the cauda equina, and about the lowest portion of the cord, there was a layer of pus. The vessels of the cord much distended with blood. The cord in the whole of the dorsal and lumbar region exceedingly soft, especially at the upper part (as high as the first dorsal), where it was almost diffuent. The gray matter was of a deep colour. There was no disease of the bones. Slight recent pleurisy on both sides. Pulmonary tissue healthy, with the exception of slight emphysema. Heart healthy. On either side of the spine, where the anterior branches of the nerves pass forward, collections of pus extended along their course for a short distance. This was the case with the fifth, sixth, seventh, eighth, ninth, tenth, and eleventh dorsal nerves. These abscesses communicated with the pus contained in the spinal canal. The lumbar nerves were not thus affected. Liver and spleen healthy. Kidneys much congested, and the cellular tissue around them œdematous. The small intestines healthy. The mucous membrane of the whole of the cæcum, and five or six inches of the transverse colon, affected with acute diphtherite. The solitary glands in the other portions swollen. Bladder distended with urine.

*Remarks.*—The extent to which paralysis occurs, in inflammation of the spinal membranes, may obviously depend not only on the amount, and seat, and character of the exudation, but also upon the presence or absence of softening or other lesion of the cord itself. In proportion as the cord is involved in the inflammatory action will the symptoms usually considered characteristic of an affection of the membranes be less and less marked, and those of paraplegia predominate. It was so in this case. Within thirty-six hours from the commencement of the disease the patient was unable to leave his bed on account of weakness in the legs, and on admission there was complete loss of motion and sensation. It was also remarkable how entirely the functions of the brain were undisturbed

throughout, contrasting, in this particular, with a large proportion of the recorded cases of acute spinal meningitis. These varieties are explained by the conditions which give rise to the disease, the nervous temperament of the patient, the degree of attendant paralysis, and the presence of actual disease in the brain itself.

The collections of pus in the course of the nerves show how the inflammatory action may be continued from the dura mater along their sheaths. There is a possibility of these purulent depôts being mistaken for the secondary abscesses of phlebitis, from which they are without difficulty distinguished by their continuity with the exudation upon the theca.

*CASE V.—Arachnitis of the cord following an injury. Paraplegia towards the end of the case.*

For many of the particulars of the following case I am indebted to my friend, Dr. Wilks.

Frederick L.—, æt. 22, a strong muscular porter at a railway station, had his neck and shoulders severely squeezed between the buffers of two carriages, on the 20th of September, 1855. He was unable to work for three or four weeks, and felt much pain in the right arm, scapular region, and down the back, especially between the seventh and tenth dorsal vertebræ. The pain was increased by any sudden twist of the body, and extended to the abdomen. About a week before he came into the hospital he was again obliged to leave his work, on account of the severity of the pain along the spine. He was admitted under the care of Dr. Addison, Feb. 6th, 1856. There was pain on pressure over the lower dorsal vertebræ, pain in the abdomen, and occasional tingling in the hands and feet. The abdomen itself was full and hard, with pain on suddenly turning the back, extending from the ribs below the umbilicus. Nothing abnormal was discoverable in the chest. The pulse was 78. Bowels regular, appetite defective. Tongue rather furred in the centre. He was treated by cupping, mercurials, and laxatives. On the 11th the pain in the back was increased. He had headache, and his nights were restless and disturbed by dreams. The shooting pain in the abdomen continued, and it was noted that the integuments were remarkably hot and dry. The pulse was 72, with a noticeable sharpness in the beat. From this date he became slightly affected by mercurial action, and was apparently improving. He left his bed for several hours in the day, without inconvenience, still, however, complaining of his former symptoms and of pain through the chest. On the 28th he had general febrile symptoms, with cough, and hurried breathing, and signs of pleurisy at the base of the left lung. The abdomen tense;—constipation. Pulse 112. Sleep disturbed by dreams, and by frequent spasmodic twitchings of the extremities. Complained very much of pain in the lumbar region, on each side of the vertebral column, and down the sacrum. On the 11th there was retention of urine. On the 13th slight delirium, and a marked decline of strength. He was scarcely able to move the legs, but the sensation

on pinching was acute. He lay supine, sinking to the foot of the bed, his arms being too weak to help him to support himself. From this date he became rapidly worse, with much cerebral oppression. The urine drawn off daily by the catheter was ammoniacal, with large deposit of phosphates. The fæces escaped involuntarily. Frequent convulsive twitchings, both of the upper and lower extremities. Breathing hurried and laborious. Tongue dry and brown. Pulse 108. On the day before his death he lay nearly insensible, frequently moaning and sighing. Pulse feeble and irregular, 90. Urine, drawn off by catheter, copious. Fæces passed involuntarily. He died on the 17th, six months from the accident.

*Sectio cadaveris.*—The head was not examined. No injury of the vertebræ or ribs discovered. Spinal canal and external surface of the dura mater healthy. The friends would not permit an examination of the whole cord. The part removed corresponded to the lower cervical and eight upper dorsal vertebræ. On opening the dura mater the arachnoid appeared remarkably thickened and flocculent, from effusion of lymph beneath it. The effusion was greatest on the posterior surface of the cord along the median line, but at the lower part of the cord it extended round to the anterior surface, and upwards for a short distance; the cord itself was not softened, nor, on repeated microscopical examination of the nervous substance at different sections, were any traces of exudation discovered. The dura mater had undergone no alteration, except that the inner layer was rather opalescent. One or two very small fibroid plates existed on the close arachnoid. The flocculent effusion covering the cord presented under the microscope the usual appearances of inflammatory exudation on serous surfaces in the stage of organization into permanent adhesions. Old adhesions over the surface of the upper lobes of both lungs. At the lower part of the left chest a circumscribed space, containing about a cupful of purulent fluid. Pulmonary tissue of both lungs stuffed with softish, yellow, miliary tubercles, equally diffused from apex to base. Heart and liver healthy. Kidneys large, the cortical portion studded with miliary tubercles. The splenic tissue similarly affected.

*Remarks.*—This case exhibits the more characteristic symptoms of pain attendant on spinal meningitis; pain in the course of the spine radiating through the trunk on any sudden twist, or other movement of the back; pain, with tingling, numbness, and twitchings, in the extremities; pain in the abdomen, with hot and dry integuments, and probably, if more carefully noted,



oscillations of temperature. Olliver considered pain having these characters as one of the most constant symptoms of spinal meningitis, but, like most symptoms, its presence is not invariable. In Case VII there was no pain in the back on movement or percussion, and the patient asserted that even the blow of a sledge hammer on the spine would not hurt him, he was so sound there. Yet the whole membranes of the cord were thickened and agglutinated by chronic inflammation. The effusion in this case was, as usual, under the close layer of arachnoid, and principally on the posterior surface of the cord, probably from gravitation. The character of the tubercular infiltration of the pulmonary tissue, and the occurrence of pleurisy with suppuration, must, as before noticed, be considered as having a probable relation to the state of the cord.

The following case, though not strictly admissible here, since at no stage was there paraplegia, is of great interest as an illustration of the apparently slight causes which may set up disease about the spine and cord. I am indebted to Dr. Wilks for the particulars of the case, and to Mr. Birkett for permission to record it.

CASE VI.—*Suppuration of the spinal membranes and formation of pelvic abscess after a blow on the back with the fist.*

Anthony P—, æt. 15, admitted into Guy's Hospital, May 15th, 1856, under the care of Mr. Birkett. He was employed with his parents in a travelling show, and was in good health until three days before admission, when, playing with another lad, he received a blow on the back from his fist. He thought little of it at the time, but afterwards, the pain becoming severe, he applied for admission into the hospital. After the application of leeches he was so much relieved that he thought of going out, but the pain soon returned more severely, and fever ensued. An abscess formed on the right side of the sacrum, which was opened, and continued to discharge, the flow of pus being increased by pressure on the abdomen. He continued daily to get worse, with much irritative fever, and severe pain in the back. During the week preceding his death he was exceedingly restless, and often delirious, and complained of *pain in all parts of the body, but particularly in the extremities*. His head was generally drawn backwards, as in tetanic opisthotonos. On one or two occasions he had loss of power over the bladder and rectum, but had no other symptoms of paraplegia, and could move freely in bed. He died June 4th, twenty-two days from the receipt of the injury.

*Post-mortem examination by Dr. WILKS.*—The external



opening at the side of the sacrum passed into a very extensive sub-peritoneal abscess, occupying the forepart of the sacrum behind the rectum, and extending over the ilia on both sides behind the psoas muscles. The bones were exposed, but not diseased. Although the abscess had discharged externally on the right side, it was most extensive on the left. It had burrowed up to the left side of the last lumbar vertebra, and through the sacro-vertebral foramen into the spinal canal. When the theca was opened, it was found to contain a quantity of greenish pus, spread over its inner surface, and over the cord itself. The dura mater, at the point indicated, was softened and destroyed, and the cauda equina was lying bathed in the pus which filled the sacral canal. The membranes of the cord were inflamed throughout their whole extent, and there was purulent effusion as high as the dorsal region. The dura mater was thickened, its inner surface had lost its smoothness and transparency, and was of a dull green colour. Pus could be pressed out from beneath the visceral arachnoid in considerable quantity. The cord itself was firm, and the microscope discovered no morbid condition. On opening the cranium, traces of acute arachnitis were found over the whole surface of the brain, greenish coloured lymph being effused into the sub-arachnoid tissue, especially at the base. The inner surface of the dura mater, around the foramen magnum and on the adjacent part of the occipital fossa, was of a greenish colour, from lymph effused upon it. Pleuræ healthy. Lungs healthy. Bronchial tubes filled with tenacious mucus. Heart normal. Lumbar and bronchial glands slightly enlarged, and containing traces of tuberculous deposit. Kidneys and liver healthy. No peritonitis nor pericarditis.

CASE VII.—PARAPLEGIA.—*Chronic inflammation of the spinal membranes. Œdema and softening of the body of the cord.*

Noah F—, æt. 46. Admitted into Guy's Hospital, under my care, June 22d, 1855. A dancing-master, of rather spare frame and nervous temperament. His general health has been good. In early life he was addicted to venereal excesses, and had gonorrhœa several times. Has taken great exercise, and often walked long distances. Can give no account of any exciting cause of his present symptoms. Twenty years ago he had gout (?), and a return of it ten years ago. After the last attack he became subject to headache, dimness of sight, double vision, pinching pains in the neck, and numbness about the mouth. He was cupped, leeches, and blistered, without benefit, and

was then ordered to the sea-side, where he soon recovered. A year ago, he noticed he could not give "the step" to his pupils so adroitly as he had been accustomed to do, but as he had no other symptom he took no notice of this, and continued to follow his profession as usual. Six months ago, the sphincters became weak, and he began to suffer from obstinate constipation. His symptoms became rather suddenly aggravated three months ago, when he found, after sitting, he was unable to stand steadily for some minutes. He now began to have rheumatic (?) pains in the right arm, soreness in the soles of the feet, and numbness in the legs, with gradually increasing and permanent weakness in them; yet he was able, until a few days before admission, to hobble about with assistance. The symptoms of paraplegia came on with frequent and very troublesome spasmodic startings in the legs, and a peculiar sense of deadness round the lower ribs. *Present condition.*—He appears prematurely aged. The cranium is well formed. The features very intelligent, but expressive of suffering. As he lies in bed he can move the legs feebly, but has no power to stand. The sensibility is diminished below the distribution of the seventh dorsal nerve, and he has a sense of constriction around the lowest ribs, extending to the spine. The inner side of the right arm and forearm feels as if "asleep," and the fingers are weak. The *excito-motor* actions are produced by the slightest touch, or by the mere shaking of the bed; and even when quite undisturbed he is greatly troubled by what appears spontaneous startings of the legs, but which are really due to the involuntary passage of the urine, at intervals, through the urethra. The legs are more or less permanently flexed. The spine is not in any way distorted. There is no tenderness on pressure over the vertebræ, nor does the application of a hot sponge give any kind of uneasiness. He says his back feels quite strong, and if struck with a hammer there, it would not hurt him. There is obstinate constipation, and when an evacuation passes he is not aware of it. The urine is acid, and without albumen. He has either complete retention, requiring the use of the catheter, or continued dribbling. Pulse 92, feeble. Tongue moist, coated with whitish fur. Frequent profuse sweats. Emaciation. A few days after admission the urine became ammoniacal, and dribbled away continually, producing excoriation of the scrotum, and a bed-sore over the left trochanter. The rigidity and flexion of the legs increased, and rest was prevented by the continual spasms of the lower extremities, which were so violent on one occasion as to jerk him off the bed on to the floor. On the 16th of July he was attacked with sickness and hiccup, and became altogether so much worse that his friends removed him home. He was visited at intervals until his death, October 21st, 1855. During the three months from his leaving the hospital he gradually emaciated. The legs became permanently drawn up, the heels to the nates, and the knees to the abdomen, the muscles flaccid and wasted. Any attempt to move them gave him great pain. The urine constantly dribbled away. The large intestines were emptied by enemata. Bed-sores formed over the trochanters and sacrum, exposing the bones to a great extent. The feet became œdematous, and a large slough formed on the heel from pressure. The tongue became dry; the mouth aphthous. He had frequent vomiting. His intellect remained quite clear, and his mind tranquil, to the end. Though sensation was diminished in the lower extremities, he retained the power of telling which toe of either foot was touched. The slightest touch of the feet or succussion of the bed, set the whole of the muscles of the lower extremities into increased contraction, and gave him great suffering. It was easy to see how an advance of disease into the cord would have greatly mitigated his miserable symptoms.

The cord only was examined post-mortem. I was assisted by my friend Dr. Habershon.

The bony canal was healthy, except a very slight prominence of the intervertebral substance at the lower part of the dorsal region, which, though unimportant in this case, was worthy of note in reference to some cases of paraplegia recorded by Mr. Key. The whole of the spinal membranes, from the lower part of the cervical region, throughout the dorsal, and to a less degree in the lumbar region, were much thickened, and adherent together. The posterior layer of the dura mater in the upper part of the dorsal region, was indurated by bony plates between its laminæ. These, examined microscopically, presented the characteristic osseous lacunæ and canaliculi, but differing from the normal bone of the skeleton in the larger and more variable size of the lacunæ, and the less numerous and delicate channelings of the canaliculi. Dr. Wilks, who, as well as myself, examined them, noticed that the lacunæ had a disposition to arrange themselves in concentric rings, being formed into parcels or systems by fibrous columns running between them. The arachnoid was quite opaque, and very thick. The pia mater also was much thickened. The body of the cord throughout the whole of the dorsal region was wasted and soft. The surface of the columns under the pia mater was translucent from granular exudation. Among the nervous tubules there was abundant granular exudation and granule cells. The continuity of the columns was nowhere interrupted. The lesions were due to chronic inflammation of the dura mater and more recent subacute inflammation of the other membranes, extending to the body of the cord. The bony plates of the dura mater were seated in the substance of the thickened membrane itself, and probably arose from the degeneration of new fibrous tissue. There were none of the opaque pearly plates so common on the arachnoid of the spine. This membrane was very thick, and its surfaces agglutinated by firm but recent adhesions.

*Remarks.*—The absence of pain and tenderness in the course of the spine was remarkable in a case where the membranes were so extensively affected; neither was there the exalted sensibility which Olivier regarded as pathognomonic of affections of the spinal membranes. The symptoms throughout



corresponded, in a great degree, with those observed in cases of tumours producing pressure on the cord, with the important exception of there being no local pain in any part of the back. There was rather numbness than exalted sensibility, and yet withal, great pain towards the end of the case, when the paralysed extremities were moved. Cruveilhier has drawn attention to this symptom of paraplegia from spinal meningitis. His conclusion, though scarcely confirmed by this case, may, perhaps, be noticed here. He states that, "in paraplegia from spinal meningitis, there is—1st. Paralysis of the cutaneous nerves, gradually and successively invading the lower extremities, the trunk, and the upper extremities; at first, limited to a portion of a limb, afterwards affecting the whole, and thence extending to another. 2d. Muscular paralysis in the first period, from pain; and muscular paralysis with anæsthesia in the second period. The muscles are painful on pressure, in voluntary or involuntary contraction, or when moved mechanically. In the first period there is voluntary power to move the muscles, if an effort be made to overcome the pain; but after a short time the pain increases so that the will is powerless over the muscle. The tenderness of the limbs is not due to exaltation of the cutaneous sensibility, as the skin is insensible, but to a painful state of the muscles themselves."

The painful state of the muscles here described did not exist in this case, nor did the pain on moving the rigid and paralysed extremities appear to arise from the muscles, but from the state of the joints, or the parts about them, due to long-continued immobility. Though a degree of anæsthesia occurred in the advanced stage of the case, the symptoms were not, as Cruveilhier states, ushered in by an affection of the nerves of sensation, but, on the contrary, of the nerves of motion; and though it is obvious there may be cases where, from the inflammation affecting the posterior roots of the nerves as they arise from the cord, alteration of cutaneous sensibility may be an early symptom, yet we can scarcely understand how it should have any such necessary law of gradual and successive invasion as that here laid down, since it is obvious this must depend upon the locality and character of the effusion, and will vary with the case.



The apparently centric spasms which affected the paralysed extremities, and occasioned this patient so much distress, were really *excito-motor*, and due to the dribbling of a few drops of urine, at short intervals, along the urethra.

As to the causes which gave rise to this extensive chronic inflammation of the membranes, I learned, after the patient's death, that he had on one occasion a very severe fall upon the back, and after that, his symptoms gradually came on. He himself attributed his paralysis to the fatigue of his occupation.

Beginning with languor and partial loss of motion, with the entire absence of local symptoms in the spine, in a man having the occupation of a dancing-master, and who had exhausted his system in many ways, his symptoms were not unlikely to be attributed to mere debility, and to be met by tonics and stimulants, instead of being combated by such means as arrest inflammation. Taken at the onset, there is reason to believe much benefit would have followed a judicious and rigid treatment.

CASE VIII.—*Paraplegic weakness of the upper and lower extremities. Wasting of the muscles. Mental confusion and delirium. Increased sensibility of the whole surface.*

Mrs. —, æt. 37, married; mother of four children. Previous to her present illness her health was impaired by an attack of cholera, and chronic diarrhoea, and probably, also, by habits of intemperance. About the end of March, 1850, she began to complain of pain in the back, wandering pains about the body, and of weakness and pain in the knees and ankles, supposed to arise from rheumatism. In a short time she was unable to walk, and the upper extremities became generally weak. The hands dropped, and hung flaccid and loose from the wrists. The muscles of the upper and lower extremities wasted, but especially of the parts most removed from the centres, as those of the thumb, the interossei of the fingers, and the muscles of the foot and calf. As the paralytic symptoms came on, there was a general change in the mind. She became cunning, more fond of drink, and inconsistent and trifling in her manner, and at times delirious. When admitted into the hospital, June 5th, 1850, there was tremor, and great mental confusion, but she retained so much consciousness as to be able to tell the number of fingers held up, and to put out the tongue when bidden, though she did it but imperfectly. On the 30th, she lay supine, sinking to the foot of the bed. Pulse 120. Respiration 36. Skin hot. Tongue dry and brown. Muttering delirium. Sufficient consciousness to partially protrude the tongue when told to do so. Both hands dropped, and useless, yet she has power to move the arms. The legs are extended; she is unable to flex them, though she can slightly move the toes, and make the muscles of the legs contract to some extent by an effort of the will. Sensation not impaired. Complains bitterly when the legs are moved,

and there is general increased susceptibility to pain over the whole surface. Speech and deglutition imperfect. Urine and feces passed involuntarily. No deafness. Pupils rather large, but contract freely on the stimulus of light. From the above date, the breathing became gradually more and more embarrassed, from the imperfect power to raise the lower ribs and to use the diaphragm. The respiration was entirely superior-thoracic, the abdomen falling in at each inspiration. There were slight twitchings of the hands, but the legs lay extended and motionless, nor could any excito-motor movement be produced by irritating the soles of the feet. She died from asphyxia, July 5th, 1850.

*Sectio cadaveris.*—The spinal cord was examined first. On laying open the theca the cerebro-spinal fluid appeared to be much increased in quantity, though still limpid. The membranes presented no obviously abnormal appearance. At the origin of the third, fourth, and fifth cervical nerves, the cord seemed, as the finger was lightly passed over it, rather softened, but a careful microscopical examination of several sections gave no evidence of any structural lesion. The gray matter was pale. Except increase of the subarachnoid fluid, and paleness of the gray substance of the convolutions, the brain was healthy. No increase of fluid in the ventricles. The phrenic nerve, and the nerves of the brachial plexus on both sides, were examined, and found healthy in their general and microscopic structure. The wasted muscles were pale and flaccid, but preserved their normal microscopic appearance. The parietes of the body, notwithstanding the great muscular atrophy, were covered with fat to the thickness of nearly an inch. The mesentery and omentum, and also the heart, loaded with fat. The right lung adherent to the parietes by old cellular adhesions, and by a deposit in these of hard, fibrous, schirroid masses. The pleura pulmonalis, on the left side, was studded with numerous small tubercles, and partially adherent by tough false membranes. The cervical glands were affected by a deposit similar to that on the pleura. Liver large and pale, from fatty degeneration. Kidneys healthy.

*Remarks.*—The origin of this case seems to have been a cachexia from previous disease and intemperance, leading to chronic cerebro-spinal meningitis and dropsy of the membranes, with atrophy of the cord and brain. The clinical history and post-mortem appearances indicate a relation to general paralysis of the insane, with this, amongst other points of difference, that the spinal centres were more affected than in that disease.

The extreme wasting of the muscles of the fore-arms and hands, and of the legs and feet, whilst the paralysis was still incomplete, led to a suspicion of primary degeneration, either of the muscular fibre, or of the nerve-trunks, but this was not confirmed by microscopical examination.

Increased membranous effusion, probably producing pressure, defective nutrition, slight softening, and paleness of the gray matter, were the only anatomical lesions to which the paralysis was attributable, and to what extent the centres of the cord had lost their functions, may be inferred, not so much from the loss of voluntary power, as from the great muscular atrophy, the total extinction of the excito-motor actions, and death from paralysis of the chest.

As an illustration of paraplegic affections, the case belongs to the class which has been termed "encephalic," or "cerebro-spinal," as distinguished from the cases which have a peripheral, or a strictly spinal origin. It includes a large number of cases of paraplegia which come on after or about the middle period of life, where at first the loss of power is not so obvious as the want of management of the muscles; the memory becomes defective; the temper irritable; the pupils inactive, and often contracted.

*CASE IX.—Paraplegic rigidity of the muscles of the upper and lower extremities from limited arachnitis of the cervical portion of the cord. The affection of the upper extremities preceding that of the lower for some months.*

Mrs. L—, æt. 33, mother of one child now fourteen years old. General health very good. Catamenia regular, up to the present time. Six months ago, having felt generally weak for a short time previous, and after a day's washing, went to bed as well as usual, but, on waking the following morning, her joints were painful but not swollen, and she was unable to move her arms. Under treatment by cupping and blisters between the shoulders, she recovered in a fortnight, and returned to her ordinary duties, having no uncomfortable symptoms but pain in the left shoulder. After another fortnight the muscles of the arms again became rigid, the affection beginning in the shoulders and extending down the arms, so that at last she was totally unable to move them. For four months after this the legs were but little affected, and she could walk until within five weeks of her admission, the arms, however, remaining quite useless from the rigidity of the muscles. On admission, under the care of Dr. Addison, February, 1854, her symptoms were as follows:—She is quite unable to move either the arms or legs, except, to a trifling degree, the left foot. The muscles of expression, of speech, and of deglutition are unaffected. Slight



anæsthesia, yet she complains of very severe pains in the knees and arms. The muscles are well nourished, and very rigid. On making efforts to pass the evacuations or urine, the whole trunk and extremities become extended, and more rigid. No pain in the spine at present, but formerly she had much in the neck, and about the back of the head. Power over the bladder diminished, but the urine can be voided at will, with some effort. Bowels constipated. Respiratory movements thoracic, heaving, and compact, not uniformly undulating. She complains much of a sense of suffocative constriction about the throat, and in speaking has no breath; nor can she cough, sneeze, or blow the nose. Speech unaffected. No affection of the nerves above the second cervical. General nutrition very good. Aspect healthy. Pulse 96. Respiration 16. Arms extended and rigid. The pain complained of in the joints depends upon the position of the limbs and the tension of the muscles, and is directly relieved, for a time, by changing their position. She has fits of shivering, which she calls hysterical; these are accompanied by increased dyspnœa. Frequent sudden spasmodic extension of legs and arms. No discoverable lesion in the bones or ligaments of the cervical portion of the spine. The pain complained of at the back of the head and neck, at the beginning of her illness, has not returned since the cupping. March 19th. No change in the symptoms. The left pupil is smaller than the right, and the vision of the left eye is imperfect, and has been so for ten months. She again complains of an aching pain in both sides of the neck, near the occiput. Pulse 120. Respiration heaving and thoracic. The excited condition of the pulse is constant, and she is subject to frequent palpitation. April 1st. General health remains good. Limbs rigid, especially the arms. She can sit upright in bed, firmly and without support, when placed in that position. The movements of the head on the atlas, and of the atlas on the axis, are free. Power over the sphincters diminished, but not lost. Pulse 90. Respiration 18. 13th. Retention of urine. May 14th. During the last few days she has had a severe pain in the head, so severe, she says, as almost to deprive her of reason. She complains bitterly of it. The sense of strangulation is very urgent. Pulse 48! Respiration 14. No delirium nor incoherence. Pupils both act on the stimulus of light, the right, as noted before, is the largest. Yesterday she was bled from the arm, without relief. On the 15th, she was delirious, and died on the morning of the 16th.

*Sectio cadaveris.*—Subcutaneous tissue very fat. Voluntary muscles generally pale but healthy, except some of the fibres of the soleus which had degenerated. Slight subarachnoid effusion on the surface of the brain. Moderate quantity of clear fluid in the lateral ventricles. Cerebrum, cerebellum, pons varolii, and central parts healthy. Floor of the fourth ventricle rather opaque, the membrane closing it thickened and bulged from the accumulation of fluid in the ventricles. The membranes of the cord thickened and completely adherent together about the origin of the third cervical nerves. Above this the adhesion extended so as to implicate the origins of the second and first cervical, and on the right side also some of the lower fibres of the origin of the pneumogastric and



lingual. The root of this latter nerve on the right side was imbedded in a mass of opaque inflammatory exudation, and a similar mass intervened between the membranes anteriorly. The roots of the whole of the cervical nerves and of the spinal accessory were matted together by old thickening of the dura mater and arachnoid. The cavity of the arachnoid was obliterated anteriorly throughout the whole of the cervical region, and posteriorly also to a somewhat less extent. The cervical enlargement and the superior part of the dorsal portion of the cord to which the membranes were adherent was softened and contained numerous granule cells. The whole of the local changes appeared to have resulted from inflammation. The yellow mass about the root of the lingual nerve consisted of dead exudation and contained the debris of inflammatory corpuscles. The other smaller mass intervening between the membranes consisted in parts of cells and nuclei, and in part of fibre cells and in one portion was organizing into distinct areolar tissue. The bones and ligaments were healthy. Lungs and heart healthy, except slight and old adhesions about the centre of the left lung, and a white patch on the surface of the right ventricle. The liver contained an excess of fat. Kidneys congested.

*Remarks.*—There are few points in paraplegia which present more difficulty than the determination at the bedside, of the causes which have given rise to the disease. In a large proportion of cases some event which, from an accident of time only, has associated itself with the accession of the more marked symptoms, is the prominent one in the mind of the patient. It was so in this case; no account could be given of a reliable cause of the inflammation, though from the anatomical conditions of the membranes post-mortem, it may be inferred that some local injury set it up. It is worthy of note that cupping and blistering at once removed the acute symptoms of the first attack, and it is probable that due care would have obviated further consequences. The case was characterised by rigidity and extension; the muscles continued to be well nourished. The principal pain complained of by the patient was from the pressure of the extremities upon each other, from the tension of the muscles, which was for the time relieved by changing their position. The upper part of the

cord was clearly indicated as the seat of the disease by the pain in the neck and the sense of constriction around the throat, as well as by the paralysis of the walls of the chest, the patient being unable to cough or sneeze. As frequently happens where the upper segments of the cord are affected, the arms were paralysed for some time before the legs. The irregularity of the pupils is probably referable to implication of the roots of the upper cervical nerves in the inflammatory thickening. Such a symptom is of interest in diagnosis, and one often misinterpreted as due to cerebral disorder when its source, as proved by modern physiology, may be entirely spinal. The acute affection of the membranes of the surface and of the ventricles of the brain which ended the case is worthy of note, as associated with the lesion of the membranes of the cord. It was also remarkable how suddenly, with the super-vention of the cerebral symptoms, the pulse, previously ranging from 90 to 120, fell to 48.

*CASE X.—Paraplegia coming on suddenly after fatigue and exposure to cold, and unattended by any derangement of the general health. Softening of the cord in the dorsal region.*

John H—, æt. 20, a healthy, florid, young man, of the middle stature, occupied as a brick-maker. On the 18th of July, 1855, he walked twenty-eight miles to look for work, and slept in a brickfield. The next day he walked thirty-two miles. The day was close and wet, and he allowed his wet clothes to dry on him, without feeling any immediate inconvenience. The following morning (July 20), he was quite well, and went out to see a cricket match. He had no stiffness in the limbs, nor pain in the back. He took his dinner as usual, about mid-day; and in the afternoon, whilst sauntering in his garden, his legs suddenly gave way under him, and he fell down. He was, however, able to get up again without assistance, and to return into the house. About two hours afterwards, he walked up stairs to his bed, feeling, as he says, all the time, “pins and needles” from the thighs to the feet. Retention of urine came on at this time, and the bowels were quite inactive. About a fortnight before his attack, he had some slight warning, in not being able, on one occasion, to pass his water for twelve hours, but from that time until the sudden accession of his symptoms in the afternoon of the 20th, he had no further inconvenience of any kind. About seven months since he had a chancre, but no secondary symptoms. Never had stricture. On admission, under the care of the surgeon (July 26) there was complete paraplegia. Involuntary twitchings and spasms of the legs towards night. Slight excito-motor movements on touching the soles, but not on irritating the skin of other parts of the feet or legs. Gradually increasing anæsthesia below the umbilicus, but nowhere complete. Bladder much distended, with slight

dribbling of urine. No priapism at the onset of the symptoms. He lies on his back, with his legs extended, and the hands under the head, with an air of entire indifference as if nothing ailed him, and says he does not feel in any way ill. Tongue clean and moist. Appetite good. No headache. Pulse 90, beat sharp. Respirations 21. Motions of lower ribs imperfect. For the last two days he has had some pain in the loins, but none previously. The spine is normal. Slight tenderness about the third and fourth lumbar vertebræ. A large bulla on the sole of the left foot from hot applications. Empl. Lyttæ lumbis; Jul. Hyd. Bichl., ʒj, ex Dec. Sarzæ, ter in die. July 28th. Complains of a sense of burning in both legs below the knees. Excito-motor action well-marked in left leg, much less in right. By straining his abdominal muscles, he can force a little urine out of the distended bladder. Urine contains mucus, and is alkaline from ammonia. Pulse 100. Respiration 28. Skin cool. He says he should be well, if only he could move his legs. *Vespere*: Had a slight rigor about mid-day. Skin hot. This rigor was probably due to a false passage made yesterday by the catheter. 29th. Return of rigors. Legs extended, and entirely paralysed, with now and then a slight involuntary jerk. The electro-contractility, even with weak currents, well marked, the electro-sensibility reduced to a perception of a faint tingling, even when the interrupted current is powerful. August 7th. Sensation of the legs slightly returning, this is most marked in the right leg. He has no sense of tightness round the waist. Excito-motor movements are now more readily produced, and follow not only when the soles of the feet, but even when the skin of the insteps, and over the legs is nipped sharply. Tongue clean. Appetite good. Skin cool. Pulse 80. Urine pale, contains mucus, and is alkaline from ammonia. 10th. Rigors, sickness, hectic. Urine highly ammoniacal. Sloughs forming over sacrum. Rapid emaciation. 18th. Rapid failure of strength. Frequent vomiting. Pulse 120. Skin clammy. Died exhausted, without delirium, August 20th, 1855.

*Sectio cadaveris*.—Body greatly emaciated. Several small superficial sloughs over the sacrum. On opening the spinal canal the sheath of the cord appeared to be more distended than usual. The inner surface of the dura mater rather opalescent. There was no abnormal adhesion nor any effusion of lymph upon the membranes; they had generally an anæmic appearance. At the middle of the dorsal region there was marked softening of the cord with slight enlargement. The softening was most marked for the extent of half an inch about the origin of the eighth dorsal nerve, but in a less degree for an inch above and below this point. There was no apparent vascularity about the part. On a transverse section the posterior columns were quite diffuent, the anterior softened but retaining their form. The gray matter was mottled by injection of its vessels. The columns were opaque white. As a general examination of the body was not permitted, the kidneys and urinary organs were removed from behind. The



kidneys were large; weight 17 ounces. The texture soft and mottled by purulent infiltration into and amongst the tubules. Mucous membrane of the pelvis congested and ecchymosed. Bladder full of purulent and ammoniacal urine. Its lining membrane inflamed and sloughing. There were three false passages from the urethra into the bladder. One of these communicated with an extensive abscess behind the bladder, and another with a smaller abscess situate to the right side of the membranous portion. On a microscopical examination of the cord the posterior columns were found to be the seat of exudation in the form of irregular masses of granules, either free or collected around softened and broken up nerve tubules, and of granule cells scattered throughout the dorsal and the lower part of the cervical region. The extent of this change was much greater than was indicated by the softening visible to the naked eye. The surface of the columns contained more exudation than the more central parts, and the white substance more than the gray. The slight mottling of the gray matter was due to injection of loops of capillary veins.

*Remarks.*—The striking feature in this case was the sudden occurrence of the paralysis without any local symptoms of pain or uneasiness about the spine, neither was there at any time that sensation of a band-like constriction round the abdomen, which is often characteristic of disease in the dorsal portion of the cord. From this and other causes, it seems probable that this symptom is more marked when the membranes and parts about the cord are affected, than when the lesion is limited to the nervous tissue only. It may have, probably, three different sources; it may arise from a subjective state of the spinal centres at the seat of disease, referred by the patient to the course of the nerves arising there; or from distension of the abdominal viscera as a result of the paralysis; or from disturbance of the muscular action of the diaphragm, and paralysis of the lower intercostals.

The absence of all constitutional symptoms during the early part of the case was remarkable. The patient had an air of entire indifference, and insisted that he felt well, but for the paralysis of the legs. If the law proposed by Duchenne had been relied on, it would have led to a grave error in diagnosis; this author having given it as a test of hysterical paralysis, that electro-



contractility is unimpaired, whilst electro-sensibility is lost, yet this was the case here, on the ninth day of the symptoms, with acute softening of the cord.

If we may conclude from the presence of granular matter and "granule-cells," that the softening was the result of an inflammatory process, the amount of solid exudation is still remarkable. It seems probable that there is some prior disturbance of nutrition of the nervous tissue, of which the traces of inflammatory action are but an after result. The pathological conceptions we may form on this point are not unimportant, for at present the theory of inflammation which obtrudes itself where acute lesions of structure occur, suggests such means of treatment as not only clinical experience but the anatomical conditions themselves show to be very doubtful. Looking at the anæmia of the membranes,—the œdema and softening of the columns,—the small amount of exudation, without any traces of plasticity, it seems probable that a supporting rather than a depletory system of treatment is most likely to favour repair, certainly the indications, both clinical and pathological, are opposed to the old empiricism, with its cupping, and blistering, and calomel.

Why the dorsal segments of the cord should be so frequently the seat of this form of softening is worth inquiry. It is a part where injuries are most felt, and probably the reparative power is less than in the lumbar or cervical regions, where the segments are more highly organized. Death resulted in this case from pelvic inflammation and abscess, probably not altogether independent of false passages made by the catheter.

CASE XI.—PARAPLEGIA.—*Subacute softening of the cord in the cervical region. Large osseous and fibroid plates on the visceral arachnoid.*

Bridget C—, æt. 30. Wife of a labourer; mother of four children; no miscarriages. Always had good health until six months ago (the fifth month of her last pregnancy), when she began to have pains in her knees and feebleness of gait. With these symptoms, there was also some pain in the neck, between the shoulders, and down the back. The hands became slightly numb, their grasp feeble, and the muscles rapidly wasted. The legs were œdematous, and it was with great difficulty she continued to walk about until her confinement. Her labour was tedious, but accomplished naturally. The child was still-born. Since her confinement, now five weeks ago, she has not left her bed. She has but slight power over the movements

of the legs. The urine has continually dribbled from her, and there has been but imperfect control over the rectum. When admitted, she was supposed to be labouring under paralysis of the bladder, from the effects of her recent labour. Two pints of highly ammoniacal urine were drawn off by the catheter. The respiration was said to be natural. Pulse 100. She was in a very helpless state from the paralytic weakness of the legs and arms. The dyspnœa and thoracic oppression were painfully urgent; she said she felt as if she wanted the space of the whole room to breathe in. She lay supine, and preferred the horizontal position, objecting, so far as she could spare breath to do so, against being placed in a more upright position. Pulse 120. Respiration 30, with noisy bronchial wheezing. Muco-purulent secretion from the conjunctivæ partially glueing the lids together. Cough feeble and ineffectual. Expectoration very difficult, muco-purulent, frothy, and viscid. Urine drawn off by the catheter high coloured and alkaline, with mucus and phosphates. Bed-sore forming over sacrum. Intellect clear. Face livid. Feet warm. No anæsthesia neither in extremities nor trunk. Sense of weight and constriction over chest. December 6th.—Symptoms of bronchitis set in this morning, with great oppression of the breathing. No expectoration. 7th.—Expectoration viscid and muco-purulent. Tongue furred. Pulse 100; very feeble. 8th.—At this date the patient was placed under my care. It was now obvious from her respiration that all the intercostals were paralysed. Instead of the chest expanding in her efforts to inspire, the walls of the thorax fell in to a marked extent, with each descent of the diaphragm. 12th.—Horizontal and supine position the same. No power to move in bed. Great dyspnœa. Respiration 44, entirely diaphragmatic. Expectoration very difficult; muco-purulent. Pulse 150. Skin hot and perspiring. Muscles of upper extremities much wasted, but she can lift the arms over the head. Slight anæsthesia of the fingers of both hands. No involuntary movements of the legs. Muscles flaccid. Abdomen distended. 14th.—Somewhat relieved of the dyspnœa by the use of sulphuric ether and brandy mixture. The expectoration lost for a few hours its puriform character and became serous. She died on the 18th, from gradual obstruction to the respiratory movements and accumulation in the bronchial tubes.

*Sectio cadaveris.*—Body moderately well nourished. Commencing bed-sore over sacrum. Head not examined. Spinal canal free from disease. No inflammatory products nor abnormal adhesions of the spinal membranes. Numerous fibroid and osseous plates, some unusually large (six lines in length by four broad), on the visceral arachnoid, mostly on that of the posterior surface of the cord, and almost limited to the dorsal and lumbar regions. Many of these contained the lacunæ and canaliculi characteristic of true osseous structure. Others had partly a fibroid, and partly a hyaline basis, with nuclei and lacunæ in it. The substance of the cord at the origin of the fifth and sixth cervical nerves was much softened. The softening principally affected the posterior columns, and the

posterior half of the left lateral column. The tissue was flocculent, and filled with granular matter and granule cells. The disorganization had most advanced at the surface of the cord, which was of a faint ochrey tint. The vessels of the pia mater at this part were full of blood. The principal softening was very much limited to the point indicated, but for three or four inches higher up granule cells were found scattered amongst the fibres of the posterior columns. Lungs healthy. Bronchi full of muco-purulent secretion. Heart and pericardium healthy. Kidneys healthy,  $9\frac{1}{2}$  ounces avoirdupois. Spleen 4 ounces. Pelvic organs healthy. Lining of bladder apparently healthy.

*Remarks.*—The cause of the softening is in this case, as it is in most others, obscure. Contrary to the statement of those pathologists, who have asserted that in acute softening of the cord the gray matter is most affected, the disorganization had in this case advanced most at the surface; and although the disease was limited to the posterior columns, yet motion was principally affected. One of the chief points of clinical interest in the case was the error in diagnosis at the early part of it, when the paralytic symptoms were attributed to injury of the pelvic nerves, and the dyspnœa and bronchitis not recognised as the effects of paralysis of the chest.

CASE XII.—PARAPLEGIA.—*Softening of the cord, principally at the lumbar termination, but extending upwards throughout the whole length of the posterior columns. Great congestion of the cauda equina. Paralysis of right third nerve from disorganization of the trunk near its origin.*

October 29th, 1855.—William L—, æt. 52. A tall man, with broad well-developed frame, twice married, and the father of a large family. Had syphilis several years ago. At the commencement of his present symptoms had enlarged testes, for which he was treated with iodide of potassium. He dates his illness from four years ago, on getting wet and fatigued, and allowing his wet clothes to remain on him, subsequently travelling to Exeter, and probably sleeping in a damp bed. Seven weeks afterwards he began to have pain in the loins, and difficulty in passing his urine, which was high coloured and ammoniacal. It was not until two months later that the first distinct symptoms of paraplegia showed themselves by weakness in the knees, and a sense of weariness in walking, which often obliged him to rest. He, however, continued to transact his business as a dye wood-cutter during the years 1852-53, and part of 1854, until at length he applied as an out-patient at the London Hospital. His paralytic symptoms were at this time attended with severe



pain running down the right leg, supposed to be sciatica. At first he was able to walk from his house in Limehouse to the hospital; but soon the legs became too weak for this, and he was much troubled, especially at night, with spasmodic retraction of them to the abdomen. The paralysis now became complete in the right leg, and he continued to suffer from the severe neuralgic pain, commencing about the last dorsal vertebra, and shooting down the leg to the sole of the foot. The left leg was occasionally the seat of the same kind of pain. Six months after becoming an out-patient of the hospital, the sphincters failed him, and large bullæ formed on the soles of the feet. He now became an in-door patient for four months, without any obvious change in his symptoms, until about a week before his admission into Guy's, when one morning on waking he found himself unable to raise the right eyelid. October 29th, 1855, he was in the following condition:—Complete paralysis of the right leg. Can flex the left thigh to a slight extent. Œdema of both feet. Slight electro-contractility of the muscles of the left leg. Electro-sensibility above the knee on this side in excess. Neither electro-contractility nor sensibility in right leg. No excito-motor action in right leg, slight twitches of left. Pain at the last dorsal vertebra extending down the legs to the soles of both feet. When the feet are roughly touched or pinched, the sensation is painful and burning. Ptosis of the right eye; paralysis also of the superior, inferior, and internal recti, with dilated pupil. Diplopia of objects to the left. Transient numbness in both hands, with slight permanent diminution of sensation in the right; no want of power in either. Tongue protruded straight. Deglutition good. He retains some power to empty the bladder; urine not albuminous, acid. Bowels inactive. No sloughs on back. No sense of constriction round the trunk. November 14th. Paralytic symptoms unchanged. During the last four days he has complained much of headache over the forehead and vertex, and the pain down the back is more intense. To-day his manner is quick and talkative, with slight delirium. The urine dribbles into the bed. Bladder distended. He complains of chilliness, and yesterday there was a perceptible coldness of the left arm and hand. Respiration normal. Pulse 84. Diarrhœa. November 24th. Has taken no food to-day. Lies in a dull and listless state, from which he can be only partially roused. Speech indistinct. Both pupils largely dilated, right inactive, left contracts on the stimulus of light. Urine drawn off by catheter, abundant, light amber colour, rather turbid from mucus, faintly alkaline. Pulse slow and labouring. Bowels inactive. November 28th. Pulse 140, feeble. Respiration 40. Skin hot, bathed in profuse perspiration. Constant twitching of mouth, and lateral oscillation of the eyes. He has been in an entirely unconscious state for the last twenty-four hours. Died at 3 p.m. He rallied from his insensibility a few minutes before death.

*Sectio cadaveris.*—Body moderately nourished. Slight œdema of right leg below the knee. No bed-sores. Only the spinal cord and brain were examined. The spinal membranes were generally very full of blood, but especially on the posterior surface of the cord, and about the lumbar enlargement and the cauda equina. The whole cord appeared to be rather small. The adhesion between the two surfaces of the arachnoid were more than usually abundant, and on the posterior surfac



of the lumbar medulla the two layers of arachnoid and the pia mater were matted together by fine cellular adhesions. At several points the dura mater was much thickened and vascular. The substance of the cord was generally soft, the greatest softening being at the lumbar enlargement, which was of a dull chocolate colour and infiltrated with granular cells. Many of the capillaries (veins) were irregularly dilated, and encrusted with oil globules. The softening and infiltration extended along the posterior columns; which examined microscopically, were found to be extensively disorganized. The focus of these changes in the cord was the lumbar enlargement and the posterior columns in the lower dorsal region, but even in the cervical segments, especially in the posterior columns, there were found a few granule cells, and scattered or irregularly aggregated oil globules, proving that the whole length of the cord was more or less implicated in the pathological changes. In the arachnoid of the lower half of the cord were many white fibroid plates and opaque granules, not unlike miliary tubercles, but smaller and less transparent. In the posterior columns, where the granule cells were most abundant, the capillary vessels were large, irregularly dilated, and encrusted with oil. The veins of the surface of the brain were distended with dark blood (death by asphyxia). There was a large excess of fluid under the arachnoid. This membrane was mottled with fatty deposits. The lateral ventricles large, containing about six drachms of clear fluid. In the centre of the right optic thalamus there was an irregular cavity, its surface lined by dilated capillary veins full of blood, and a soft flocculent tissue, containing oil globules and granule cells; the whole of a dull ash colour, without any tinge of blood pigment, and due to advancing ulcerative absorption of the tissue. The surrounding brain substance had a "worm-eaten" appearance, and presented all the stages of decay. At the origin of the third nerve, on the right side, the pia mater was much thickened, and infiltrated with old plastic matter, becoming fibrous and vascular, and containing in it degenerated nuclei, granule cells and oil globules. The trunk of the nerve was slightly enlarged and tough, and had a yellowish semi-translucent appearance. Under the microscope it was seen to be converted into a fibrous cord, with scarcely a trace of nerve tubule. The substance

of the crus beneath was healthy. The opposite nerve was normal.

*Remarks.*—Dissipation, and the cachexia resulting from syphilis and its treatment, were probably the predisposing causes in this case, which needed only the vicissitudes of our climate to give rise to chronic lesion of the cord. The severe neuralgic symptoms, supposed to be ordinary sciatica, which attended the invasion of the paralysis in the right leg, was probably due to venous congestion of the nerves of the cauda equina, which was remarkable in this case. In support of such an opinion, I may mention having found in other instances varicose and enlarged venules in the trunks of neuralgic nerves. The diffused character of the lesion was indicated by the absence of any distinct horizontal line limiting the paralysis, the whole cord being in some degree implicated in the pathological process. The occurrence of ptosis, from thickening of the pia mater at the root of the third nerve, and the infiltration of the nerve trunk with inflammatory exudation, deserve notice, as associated with the changes in the spinal membranes. The ulcerative softening and destruction of the right thalamus may explain some cases of paraplegia complicated with amaurosis. The mode of death by subarachnoid and ventricular effusion corresponded to the chronic inflammatory changes in the spinal membranes.

CASE XIII.—*Acute paraplegia ; softening of cord ; fatty degeneration of the inter-vertebral substance ; fibrous plates on arachnoid.*

Mrs. G—, æt. 33, wife of a house-painter ; mother of one child, and now ten weeks pregnant with a second. Up to the time of this illness always had good health, though apparently of rather a delicate constitution. Complexion fair. About mid-day, on Monday, January 12th, 1855, whilst engaged in her domestic duties, she was suddenly seized with severe pain in the back, making her feel sick and faint. This lasted for half an hour, and then entirely left her. In the afternoon she went out, carrying her child, and returned home, feeling as well as usual, except being fatigued, which she attributed to having a cold. The next day, after passing a good night, she went about her household work, feeling very well until noon, when almost suddenly she became paraplegic. There was complete loss of sensation as high as the waist, as well as of voluntary movement, and entire loss of control over the sphincters. She was admitted under the care of Dr. Barlow February 23d, 1855.

Since her seizure there has been a gradual return of sensibility, and of some power over the right foot and ankle. There are several sloughs on the feet, from the application of hot water, and much larger ones at the lower part of the spine, and over the hips. The face is pale, expression anxious. Pulse 120. Respiration 28, performed by the diaphragm and the five upper ribs, the lower intercostals being paralysed. The spine is straight and free from any irregularity. No tenderness at any spot. The urine withdrawn by the catheter is acid; sp. gr. 1025. On questioning her, she denies having any feeling of constriction around the chest or abdomen, but complains of a slight sense of weight at the sternum. No distension of the abdomen. Liver extends two inches below the ribs. February 27th. Her nights are restless. Continued hectic symptoms. Face now flushed. Tongue furred and dry. No headache. No delirium. Pupils rather contracted. Can move the right foot and ankle slightly. Left leg quite immoveable. Sensation perfect. Yesterday had a rigor, repeated at bed time. March 3d. Sloughs on back and hips extending. No complaint of pain. Tongue dry and brown. Stiffness between the shoulders, and aching pains down the back of the arms. Occasional spasmodic twitchings in the left leg, otherwise both are motionless. *Supposed* diarrhoea due to the constant passage of semi-solid feces through the paralysed sphincter. 7th. Great emaciation. Pulse 130. Respiration quickened. Slight cough; imperfect from the paralysis of the lower intercostals. Muco-purulent expectoration. The passage of the urine and feces can still be felt, but is quite involuntary. 10th. Rapid decline of strength. Upper part of the trunk and arms perspiring. Skin of lower extremities dry and harsh. The paralysis of motion remains as before. Sensation throughout the paralysed parts nearly perfect. The smallest point can be felt, and the distance between two points appreciated as in health; yet the acuteness of the pain from pinching the skin is diminished. The symptoms were noted from day to day, but did not vary in any essential respect. She died on the 18th. The pulse was imperceptible at the wrist for many hours before death. No delirium or incoherence throughout the whole course of the disease, which lasted nine weeks and three days.

*Sectio cadaveris.*—Body much emaciated, with extensive sloughs, as described in the report. On removing the cord with its membranes from the canal, the parts were healthy, except a small amount of opaque, cheesy matter, oozing from the fifth intervertebral substance. The dura mater was healthy. The arachnoid was free from adhesions, and every where normal, with the exception of many ossific plates scattered over its visceral layer, especially about the cauda-equina. The substance of the cord was much softened, from the tenth dorsal vertebræ upwards for six inches and a half. The posterior columns were diffuent, the anterior were continuous. The left column was more softened than the right. The lower section of the softened part, for about two inches, was of a dull pink colour, and the vessels of the gray matter much injected. There was no trace of effused blood. Amongst the softened



tissue of this part, there were a few granule cells of various sizes, and here and there an exudation cell having the ordinary appearance; but above and below this point, the columns, though soft, gave no traces of corpuscular exudation, the texture being simply loosened. The amount of exudation, even at the point of greatest softening, must have been small, as the cord was not sensibly swollen. No trace of plastic exudation on the membranes. On making a section of the bodies of the vertebræ, the intervertebral substance of the fifth, sixth, and seventh was found softened, and in part opaque, from fatty degeneration of the fibrous stroma and the cartilage cells. The degeneration was most advanced in the fifth intervertebral substance, where the adjacent portion of the bone was becoming absorbed, and the fibrous structure of the posterior common ligament had in part yielded, and allowed some of the debris of the intervertebral substance to be squeezed into the canal, and so to injure the cord. This was the yellow matter seen on removing the cord and its membranes, and at first supposed to be strumous exudation. The lungs were free from all traces of tubercle. The lower lobes in a state of reddish gray consolidation, easily lacerable. The bronchial membrane injected and granular, and covered with tenacious puriform mucus. Liver weighed four pounds avoirdupois; tissue pale, fatty. Kidneys soft; tissue coarse; weight ten ounces avoirdupois. Mucous membrane of bladder inflamed. Sloughs extending through the anterior wall to the sheath of the rectus, and posteriorly destroying the vagina. The os uteri sloughing, and a small ovum, well-formed, protruding. The mucous membrane of the small and large intestines much congested. In the stomach, at the larger curvature, near the fundus, were several ulcers of the size of a sixpence; the black sloughs of the mucous membrane being still adherent.

*Remarks.*—The exciting cause of the acute softening of the cord in this case was mechanical injury, resulting from the giving way of the posterior common ligament, and the escape of the debris of the degenerated intervertebral substance into the canal. The sudden pain in the back, with sickness and faintness, felt by the patient on the day previous to the paralysis, probably depended upon this rupture. It was thought when the canal was first opened, that the lesion of the inter-



vertebral substance was due to scrofulous deposit; but that opinion was not supported by a further examination. It appeared to be only a form of atrophy, leading to opacity and fatty degeneration of the texture. As it was but a small spot of the posterior surface of the intervertebral substance which was affected, there was no displacement of the bones. The recovery of sensation even whilst the case was progressing to a fatal termination is of interest, as bearing upon prognosis. The same is observed in hemiplegia, from softening or effusion of blood into the corpus striatum, or thalamus opticus, the loss of sensation accompanying the injury to the nervous centres, being after a few days recovered, though in other respects the symptoms may have undergone no favorable change. The subject has also a further interest in reference to the physiology of the sensitive functions of the cord, especially, as it will be observed, that the posterior columns were broken down, and only the anterior continuous. The cause of death was exhaustion, from the unusually extensive sloughing of the pelvic viscera, and of the skin over the sacrum.

The amount of exudation into the cord was, as usual, very small, and except at the part principally affected, the tissue was simply loosened, and œdematous. Some of the granular bodies seen under the microscope, were formed by the aggregation of granular matter around broken nerve tubules, others had the more common origin from degenerated exudation corpuscles. Whether such a lesion as this, apart from the chronic disease of the surrounding structures, is remediable, is very doubtful; but, as observed in the preceding case, we should expect less from the use of calomel than from those means which favour nutrition.

CASE XIV.—*Paraplegia preceded by symptoms of colic. Sudden loss of power and sensation in the upper extremities; partial recovery for some months; relapse. General and slight softening of the whole cord. Traces of inflammatory exudation discovered in the cervical portion, and in the medulla oblongata.*

Many of the particulars of this case were collected for me by my friend Mr. Edmund Galton.

Esther J—, æt. 32, a stout, leucophlegmatic woman, a widow, never had robust

health, and, as a girl, was subject to severe headaches, and at times to hysteria, and also to painful and irregular menstruation and palpitation. Had, according to her account, two attacks of pleurisy eight years ago, and soon after was in St. Thomas's Hospital for rheumatism. Eighteen months ago, had symptoms of colic, attended with giddiness and slight mental confusion; and about that time, on waking one morning, found she had lost to a great extent the power of motion and sensation in both upper extremities. The hearing became at the same time dull, and her memory impaired. She gradually recovered the use of the arms, and continued in her usual health (though not able to walk up and down stairs, and occasionally having difficulty in breathing), until two months ago, when she began to have pain in the back between the shoulders, increased difficulty of breathing, pains in the limbs, with formication in the fingers, and pain in the left side and abdomen. A fortnight after this aggravation of her symptoms the power of the upper extremities became again much impaired, the wrists dropped, and the hands became numb. The legs were less affected; she was able to stand, but not to rise from her seat without assistance. The loss of power was rather more marked on the left side than on the right. The sense of taste was lost. On admission into the hospital, August 22, 1855, the following note was made of her condition. Sensation in the left arm perfect, as far as the elbow, below it is gradually lessened, and entirely lost in the fingers. The motion of the shoulder joint unimpaired, but attended with pain in the back. Power of extending the elbow joint very imperfect, wrists dropped. Right arm similarly, but less affected. Can move the legs in any direction in bed, but is not able to stand without support. No affection of sensation. Loss of taste. Complains of pain in the back, passing over the shoulders. Tenderness on pressure over the lower part of the cervical, and upper part of the dorsal region. No abnormal condition of the spine discoverable. Severe griping pains in abdomen. Obstinate constipation. Dyspnœa, cough, and constriction across the chest, with inability to expectorate. Slight bronchial râles, respiratory sounds otherwise normal. Power to empty the bladder remains. Urine acid, high coloured. Frequent cold perspirations, followed by flushing heats. Sleeplessness, despondency, globus, and other hysterical symptoms. There was no marked change until September 9th; she was very desponding, and often expressed a wish to die. Had day by day various nervous symptoms of an hysterical character. Bowels obstinately constipated, great pain in the abdomen. Slight traces of blue line on the gums, which, with her other symptoms, and the dropping of the wrists, favoured an opinion of lead poisoning. The dyspnœa, cough, and inability to expectorate, with a sense of suffocation, continued to distress her very much. On the 9th, all the paralytic symptoms were in a few hours increased, with intense pain between the shoulders, and across the chest. The urine was passed naturally. Pulse weak and frequent. On the 11th, she was universally paralysed. There was frequent cough with inability to expectorate, and an increased sense of suffocation. Cardiac sounds very feeble. Paralysis of the respiratory muscles gradually increased. The larynx soon became involved. She was neither able to speak nor swallow, but remained perfectly sensible until her death at midday.

*Sectio cadaveris.*—Body well developed and stout. No sloughs nor abrasions on the back. Integuments and internal organs generally congested from the mode of death. Cere-

brum, cerebellum, crura cerebri, and pons varolii healthy. Medulla oblongata softened. Under the microscope there was seen, here and there, an exudation cell amongst the loosened tubular structure. The spine and its membranes were healthy. The cord softer than usual throughout; but it was only after examining many parts, that any trace of exudation was discovered, and that only in the cervical region. Without repeated examination this would have been overlooked, both in the medulla and in the cord. The viscera, including the kidneys and bladder, healthy, with the exception of recent congestion.

*Remarks.*—The morbid anatomy of this case is of great interest, as elucidating those recorded instances of paraplegia where no lesion of the cord was observed. It was only by great patience, that the microscope discovered any traces of inflammatory exudation, but these, though slight in amount, were distinct and decisive. It is probable the paralysis was rather due to the arrest or perversion of the normal processes of nutrition than to the mere mechanical effects of the exudation.

The early symptoms were vague, and thought by some to be hysterical, thus affording another proof that it is not in the symptoms themselves, taken individually, but in their course and grouping, that the true basis of the diagnosis lies.

Whether the paralysis was the effect of lead, as was supposed from the traces of a blue line on the gums, the dropping of the wrists, and the colic and constipation, is doubtful; as the anæmic and icterode tinge of the surface and conjunctivæ, so characteristic of lead poisoning, was wanting; neither do the affections from lead take such a course to a fatal termination. The remarks on the obscurity of causation apply to this, as to most cases of softening.

*CASE XV.—Paraplegia commencing by paralysis of the right arm, and referred to an injury of the hand. An undefined nuclear growth in the cervical region of the cord, and a similar degeneration of the gray matter throughout.*

Abraham C—, æt. 23, stoker on board a steamboat, of intemperate habits, but has had pretty good health. Has occasionally been in pugilistic encounters, and received many blows on the head and forehead, but the most severe was about five years ago,



when he was struck unexpectedly by another man's fist on the side of the neck, near the articulation of the skull with the vertebral column. Since that, he has occasionally had difficulty in deglutition, particularly of fluids, which would be expelled through the nose. For the last year he has had a choking sensation, and, at times, difficulty in passing water. He attributes the weakness of his right arm to a blow which he received on the back of the hand, eighteen months ago, by the falling of a piece of iron. This accident kept him from his work for six weeks, but the wound healed without any extension of inflammation up the arm. As he recovered, he noticed a want of power in the ring and little fingers, and the whole arm, from the shoulder, became wasted and weak. He continued to work with his left arm for three months longer; but about the beginning of the year 1850, he began to suffer from what he terms 'bile,' that is, frequent vomiting, unattended by any pain in the head or giddiness. These returns of vomiting continued for four months, and then, as they subsided, there was increased difficulty of deglutition, and both legs became weak, the left first, and to the greatest degree. In the autumn he improved, and was able to walk about, but the bladder was so far paralysed, that he needed the catheter to be passed for several weeks. The improvement was only of short duration. On his admission into Guy's, June 5th, 1851, under the care of my colleague, Dr. Barlow, the right arm was completely paralysed at the shoulder-joint, and there was great wasting of the muscles, only slight power of moving the fingers remained. There was anæsthesia increasing towards the hand, most marked in the branches of the ulnar nerve. No actual paralysis of the left arm, but the muscles flaccid and weak. He had pains running over the back of the head. He could move the legs slightly. Sensation impaired as high as the hips. No deformity of the spine, nor tenderness on percussion. No sense of constriction at any part of the trunk. Vision somewhat impaired. Urine and feces often passed involuntarily. Pulse 90. Tongue clean and pale. He improved, by rest and by the use of electricity, so far that, in October, he could support himself and walk without help, though the gait was very vacillating, from want of power to direct the muscles. No numbness remained in the legs. The right arm continued in the same state as on admission. The left was weak, and at times he had cramp in the muscles, and involuntary closure of the hand. The sphincters partially paralysed. Aspect pale and emaciated; the whole muscular system much atrophied. He continued in the hospital until June, 1852, his symptoms fluctuating between improvement and relapse. He could walk about the ward, by the aid of a stick, with a feeble gait, his right arm hanging loosely, supported only by the ligaments of the shoulder-joint. On leaving the hospital, he went to Dover, but returned, and was readmitted in October, 1852. In a few weeks the left arm was quite paralysed, and he lost the little remaining power over the legs and sphincters, and became universally paraplegic. He often complained of a sharp pain in the back of the head and in the upper part of the neck. On 19th January, 1853, bronchitis came on, from exposure in moving him from one ward to another; though trifling in amount, the distress occasioned by it was inexpressible, from the paralysis of the intercostals. A remission of his chest-symptoms occurred until March 14th, when they again became aggravated. His distress was indescribable. Constant ineffectual efforts to expectorate. Pulse rapid, 120. Respiration 36. Face congested. Complete paralysis of the extremities and walls of the chest, and general anæsthesia, yet great pain when the body or limbs were roughly handled. Frequent spasms in the legs; arms not so affected. Urine con-



stantly dribbling. Slight abrasion of the skin over the sacrum, but no sloughs occurred throughout his illness. His miserable existence was drawn out until April 12th, 1853.

*Sectio cadaveris.*—Remarkable atrophy of the whole muscular system, and of the tissues generally. Slight abrasion of the skin over the prominent part of the sacrum. No slough. Diffused tubercular masses and scattered tubercles through the upper lobes of both lungs. Dilatation of the bronchial tubes; their lining deeply injected. Contents purulent. Heart healthy. Hepatic tissue congested and fatty. Kidneys healthy. Pia mater and brain tissue rather watery. On removing the arches of the vertebræ the whole cord appeared to be large and swollen; in the cervical region the theca was evidently distended by it. There was no affection of the bones or ligaments. On laying open the theca there was a general enlargement of the cervical portion of the cord, which, on transverse section, had an unusual appearance. The columns had a yellowish tint, and were distended by a soft vascular translucent growth, parts of which were firmer and opaque yellow, (dead?) This growth was not defined, but passed insensibly into the degenerated gray matter (Plate IV, figs. 3, 4, 5, and 6), which from the floor of the fourth ventricle to the filum terminale was pale and swollen, and had much the physical character and consistence of thick boiled starch. This soft starch-like substance, under the microscope, was seen to consist of round, oval, and elongated granular nuclei, imbedded in a slimy blastema, (Plate V, fig. 3,  $\alpha$ , and  $\gamma$ ). At the filum terminale, where the more normal characters of the gray matter were preserved, these nuclei were scattered amongst the softened tubercles with exudation-cells, ( $\beta$ ). The vascular growth in the cervical region, consisted of degenerated nervous texture, nuclei and nucleated cells, as in the fibro-plastic growths. The opaque part was little else than granular matter and oil globules, ( $\delta$ ). There was no lesion of the membranes of the cord, nor was the continuity of the columns destroyed, though in the cervical region they were spread out and slightly softened in parts. The nerves arising from the cord in the cervical and lumbar regions, examined microscopically, had the normal structure.

*Remarks.*—The limitation of the paralysis at its commence-

ment to the right-arm, and the preponderating affection of the muscles of the shoulder-joint, are points in the history of this case of great interest. Taken together with the injury to the hand, to which the patient attributed his symptoms, they led to an opinion that the case was one of peripheral paralysis, but such an inference was not supported by the history of the case, nor by the post-mortem appearances of the cord. The slight affection of the muscles of deglutition, the sense of choking, and the occasional loss of power over the bladder, connected the blow at the upper part of the spine with the lesion in the cord, whilst the peripheral origin of the malady was entirely negatived by the normal microscopic structure of the nerve trunks. The general atrophy of the muscles of the extremities in the progress of the case before the more distinct symptoms of paralysis occurred, is deserving of special note, as bearing upon the theory of progressive muscular atrophy, many examples of which have no doubt had, contrary to the opinion of those who recorded them, a spinal rather than a muscular origin. The atrophy of the muscles of the right shoulder, whilst those of the fore-arm still retained some power, elucidates the seat of the paralyzing lesion in some cases of infantile paralysis of the shoulder occurring during dentition. It has been doubted whether the lesions alluded to, have a cerebral or a spinal origin, but their occurrence without any cerebral symptoms, the occasional affection of both arms or of all the extremities, and the actual observation of a limited spot of ochrey discoloration in the cord, as in one case examined by Cruveilhier, concur with the collateral evidence here afforded in proving a spinal origin of this form of paralysis. There is nothing in practical medicine more fallacious than hastily inferring a negative, from negative evidence, as was proved in this case. The absence of pain on percussing the spine, and the positive account given by the patient that his paralytic symptoms were the result of the injury to the hand, led to the conclusion that no lesion of an active kind was going on in the cord, yet we can have no doubt that the contrary was the fact. The least consideration will show that if the ligaments and bones be healthy, no amount of pressure or percussion made in the usual way of a clinical examination can much affect the structure of the cord itself, and that we can base no inference

upon the negative evidence so afforded. There is a minor symptom in this case deserving of notice: I allude to the impairment of vision. This may be associated, from different causes, with paraplegia. Here it was probably referable to the changes in the cervical portion of the cord itself, since it has been clearly shown by experiments on animals that the condition of the eye is at once affected by injuries to the roots of the cervical nerves. I have had occasion to notice this in preceding cases.

Vomiting as an early symptom of disease of the cervical portion of the cord, occurred in this case, and was probably dependent upon the origin and connection of the phrenic nerves; in another case, as we have seen, the symptoms set in with an irritating cough. I remember an obstinate case of hiccup, which having resisted other treatment, yielded at once to blisters on either side of the cervical portion of the spine, over the origin of the phrenic nerves. The character of the local changes in the gray matter was peculiar, and probably depended partly upon degeneration of the normal structure, and partly on a neoplastic formation of the simplest kind. In the cervical region, where the disease first commenced, this had progressed to the greatest extent, making an approach to the development of a tumour, but not separated by any line of demarcation from the other parts of the gray matter which had undergone a similar, but less advanced, change. The existence of tubercles in the pulmonary tissue, can hardly, in cases like this, where young persons have been long bedridden, be regarded as an index of a previous scrofulous habit, since it is more than probable, at least in some of the cases, that the tubercular diathesis was induced by the unfavorable circumstances to which they were subjected.

CASE XVI.—*Paraplegia. Early symptoms referred to rheumatism and phthisis. Induration of the cord at the cervical enlargement; softening of the dorsal segments adjacent.*

For the following case I am indebted to my friend Mr. Bradley, who also kindly sent me the cord for examination. The patient was an inmate of the Model Prison.

W. P.—, æt. 29, a single man, native of Devon, employed as a shepherd, of healthy appearance and florid complexion. His health had always been good previous to



the 25th of January, 1850, when he began to complain of pains, which he attributed to having caught cold in a bath, some days before. These pains at first occupied the left shoulder, particularly the scapula and the deltoid, but subsequently extended down the arm to the fingers, and at times wandered into the leg of the same side. From the description given of the pains, and from the absence of other symptoms, either local or general, the case was regarded as rheumatism. During a period of two months various remedies were in turn prescribed, but without any satisfactory result. The only one that afforded any relief was morphia. By this time, the pain was localized in the shoulder-blade, and though not paroxysmal, yet, from its severity, it appeared to be neuralgic. Symptoms of phthisis, also, now began to show themselves. There was great emaciation and muscular debility. Pulse 120, and weak. Profuse nocturnal sweats, especially about the head and chest. Dry cough. Chest everywhere resonant; respiratory murmur at the left apex impaired. Constipation. By the 7th of April the weakness of the lower extremities had increased, so that he was unable to stand. There was imperfect control over the sphincter ani, and the urine was retained. That drawn off by the catheter was clear, and faintly alkaline. On the 8th, the urine was ammoniacal. Motions passed unconsciously. Voluntary movement of lower extremities lost. Sensation impaired from fourth rib downwards. Upper extremities unaffected. Intellect unimpaired. No tenderness on percussing the spinal column. Appetite unimpaired. Sleep sound. No complaint of pain. The treatment consisted in the exhibition of calomel in grain doses, to affect the system. Blisters, with mercurial dressing, to the lower portion of the cervical region of the spine, and between the scapulæ. On the 18th, his condition was much the same as on the 8th, except that sensation in the lower extremities was improved. There were cramps and flying pains in the limbs, with spasmodic movements of the muscles of the upper extremities. The perspirations were profuse. Gums affected by mercury. May 1st. A seton was inserted on both sides of the spine, at the nucha. On the 13th, the cough and expectoration had ceased. Sensation had returned in the legs; he could retain his motions, and was conscious of the passage of the urine. On the 23d, the setons were removed, and, as he complained that the involuntary spasms of the limbs prevented sleep, he was ordered half a grain of morphia every night. On June 3d, he had lost power in his hands, and there was loss of sensation in the ulnar side of the right hand and back of the arm. The paralysis of the lower extremities continued, but sensation was restored, and he could retain the urine and feces. Subsequently the pain and spasms of the lower extremities were very distressing, and he complained of pain about the third and fourth dorsal vertebræ, increased by percussion. On the 20th, bronchitis set in, attended with great difficulty in expectoration. He gradually sank on the 29th.

*Sectio cadaveris.*—Body greatly emaciated. Brain and its membranes healthy. Membranes of the cord healthy. At the cervical enlargement, the cord was indurated to the extent of an inch. On section at this point the columns had a greenish yellow tint, and were of an almost horny hardness; below this part, for three or four inches, there was marked softening, the columns being nearly diffuent. Kidneys large, pelves and ureters dilated. Abdominal viscera healthy. Recent pleuro-



pneumonia at the bases of both lungs. The apices adherent, the pulmonary tissue indurated, and containing several small vomicæ (?) Bronchial tubes generally dilated, and containing muco-purulent secretion.

*Remarks.*—I have to regret that a microscopical examination was not made of the indurated portion of the cord in this case. The induration was uniform, and the cord somewhat swollen, as if from fibrinous infiltration of its textures. The chief clinical interest of the case was the obscurity of its early symptoms. For a period of two months pains, supposed to be rheumatic or neuralgia, were the only complaint, and it was even suspected that the patient, under the circumstances, might be feigning illness. The next phase was great muscular debility, rapid emaciation, dry cough, and profuse sweats. As the chest was resonant throughout, the patient was supposed to be labouring under diffused tubercular disease of the lungs, though there can be no doubt the symptoms had, as in a previous case (Case I), a spinal origin. It was not until imperfect control over the sphincters roused attention, that the spinal disease was suspected. It will be observed that after the first shock of the onset of the more marked paralytic symptoms, sensation slowly returned, as in a previous case. The spinal membranes were healthy, yet it will be observed that the early symptoms were pain, radiating in the course of the nerve trunks, and as the paraplegia became more marked, the patient was greatly distressed by painful spasmodic contractions of the legs. There was no account in the history of the case, of the exciting cause of the malady.

## PLATE IV.

### ILLUSTRATING DR. GULL'S CASES OF PARAPLEGIA.

- Fig. 1. A vascular fibro-plastic tumour, situate on the posterior surface of the cord in the lower part of the dorsal region under the close arachnoid (Case 3.)
- „ 2. A vascular fibro-nuclear tumour growing from the inner surface of the dura mater on the anterior part of the cord in the upper part of the dorsal region: the cord pushed backwards and compressed (Case 2.)

The artist has by mistake inverted the drawing.

Figs. 3, 4, 5, 6. Sections of the cord in Case 15, showing the enlargement (6) in the cervical region from the development of a soft vascular nuclear growth in the gray matter. The sections 5, 4, 3, are intended to show the same in a less degree in the other parts of the cord lower down.

Fig 1

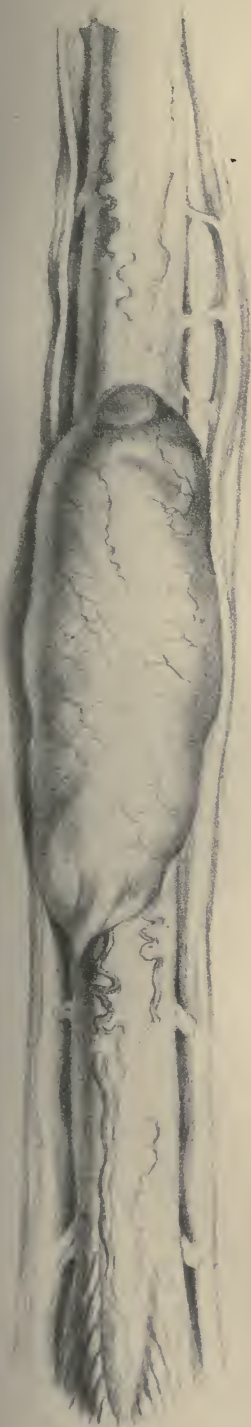


Fig 3.



Fig 4



Fig 5.



Fig 6.



Fig 2.



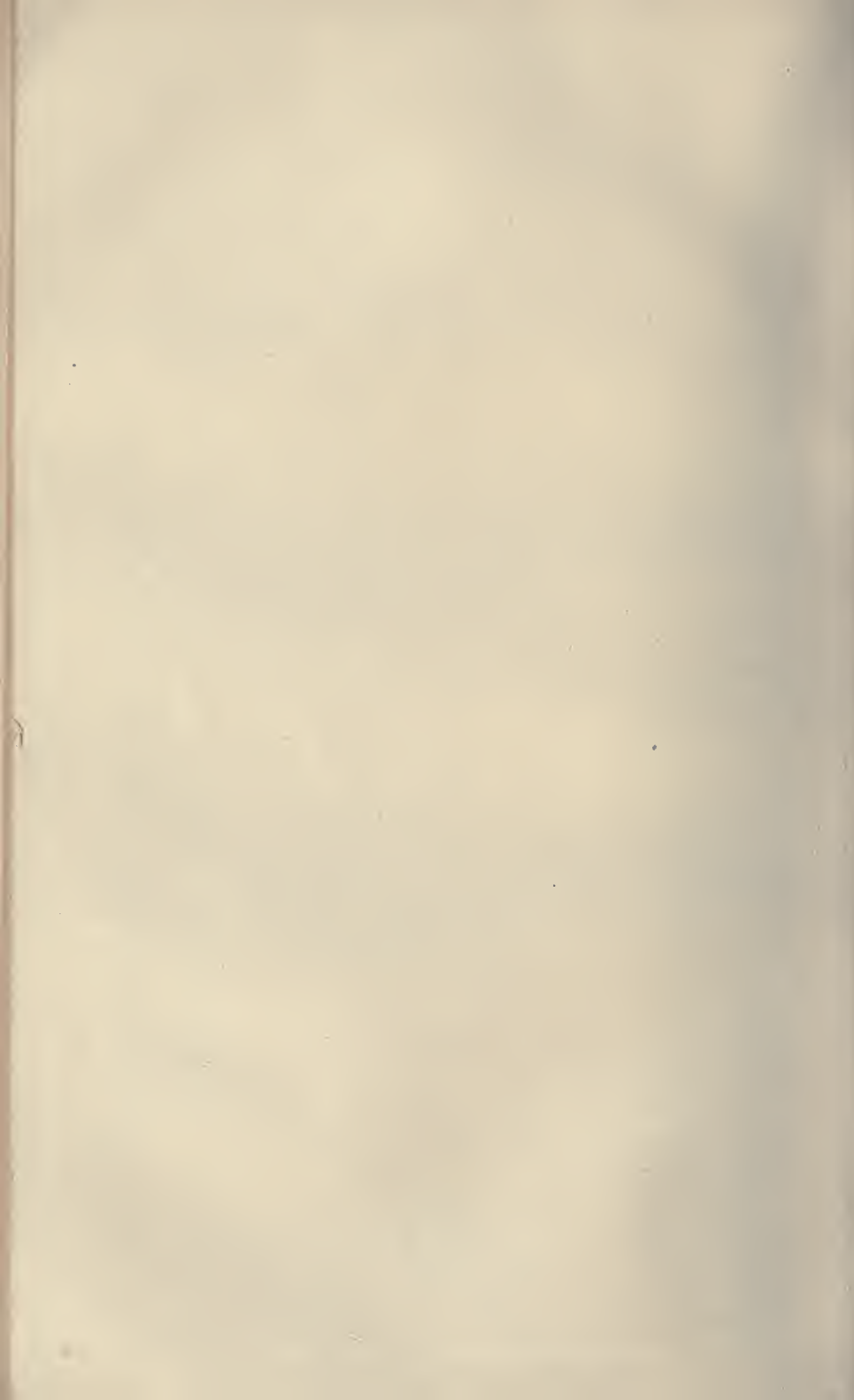






PLATE V.

Figures 1—3 illustrate Dr. Gull's cases of paraplegia.

Fig. 1 represents the microscopic appearances of the tumour in Case II.

*a* and *β*. Compacted and free nuclei with granular matter.

*γ*. Nuclei with granular matter and a cell wall around.

*δ*. Delicate granular fibrils with nuclei.

„ 2 represents the microscopic appearances of the tumour in CASE III.

*a*. Free nuclei, round and oval.

*β*. Fibrous tissue, inclosing nuclei.

„ 3 represents the microscopic appearances of the spinal cord in Case XV.

*a* and *γ*. Nuclei in a slimy blastema.

*β*. Degenerated nervous tissue with free nuclei and degenerated exudation cells.

*δ*. Oily particles from opaque yellow mass.

The above 3 figures are magnified about 240 diameters.

Figs. 4 and 5 exhibit Dr. Wilks's drawing of the cells from myeloid growths.

Fig. 4. The many-nucleated cells from the myeloid tumour of the scapula, described at p. 1.

„ 5. Similar cells from the epulis, described at p. 5.

„ 6. Many-nucleated cells from Peyer's and mesenteric glands in the case of Fever, marked No. 3, p. 139. Similar cells were also found in cases Nos. 8 and 9.

The above 3 figures are magnified about 240 diameters.



Fig. 1.



Fig. 2.

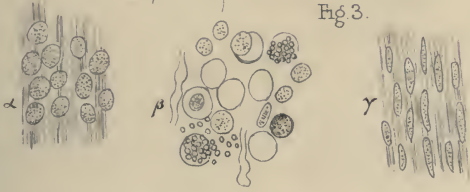


Fig. 3.

West, M.D. del.



Fig. 4.



Fig. 5.



Fig. 6.







ON THE  
PARASITICAL VEGETABLE NATURE  
OF  
PITYRIASIS VERSICOLOR,

(*Microsporon Furfur.*, ROBIN.)

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BY WILLIAM GULL, M.D.

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IN 1846, Eichstedt found a fungus growing amongst the epithelial scales in pityriasis versicolor. The discovery was confirmed during the following year, by Slyter, in a tract 'De Vegetabilibus organismi animalis parasitis ac de novo epiphyto in Pityriasi versicolori obvio.'

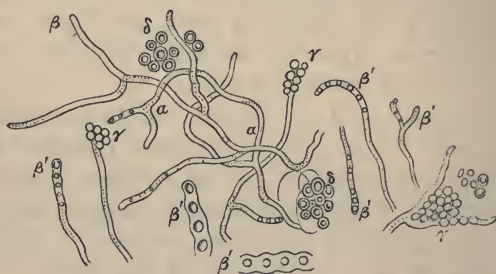
In this country, and in France, the subject still seems to remain in doubt. Robin, in his elaborate treatise 'Des Vegetaux parasites qui croissent sur les animaux vivants,' details the observations of the above-named authors, but adds in a note "Je n'ai pu verifier moi-même ces faits." Wilson, in an edition of his work on 'Diseases of the Skin,' so recent as 1851, says, "Dr. Gustav Simon places chloasma (*Pityriasis versicolor*) in his sixth group of diseases of the skin, which he entitles *Parasites*, considering this eruption as depending, like favus, sycosis, and alopecia circumscripta, upon the presence of a parasitical vegetable fungus. I do not agree with him in this opinion, and have failed to discover any vegetable organisms although I have searched for them with care.

In an edition of Cazenave's lectures, published in 1853, there are the following remarks: "Envisagé dans son ensemble le pityriasis est une affection complexe (*sic*) dont le caractère intime est exprimé par la double existence d'une

secretion anormale de la matière épidermique et d'une lésion de secretion de la matière colorante."

With the subject in this uncertain state, it will not be superfluous to record any observations which may bear upon it. In the several cases where I have looked for the fungus in pityriasis versicolor, I have always found its sporules amongst the scales of the epidermis; but until recently, I have failed to detect the ramifying branches of the mycelium itself. This failure I now know to have arisen from want of care in manipulation. The description of the fungus given by Robin,<sup>1</sup> agrees with what I have observed, except the rarity and difficulty of making out the terminations of the filaments, which I have not found. It will be seen from the subjoined sketch, that the filaments sometimes end simply, at others, by a slight enlargement, as a sporangium, with the sporules in a linear series, and at others form a receptacle with the sporules more or less regularly disposed upon them.

*Sketch of the Microsporon furfur.*



*a a.* Mycelium. The filaments  $\frac{1}{8000}$  to  $\frac{1}{10000}$  inch.

*β.* Simple termination.

*β' β'.* Terminations containing sporules.

*γγ.* Terminations as receptacles.

*δ δ.* Sporules free,  $\frac{1}{4000}$  to  $\frac{1}{5000}$  inch.

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<sup>1</sup> Trichomata (fila) in squamis epithelialibus sita, numquam etiam earum marginem excedentia, multipliciter torta et inter se nexa ut raro fili finis cujusdam certo cognosci queat; simplicibus parallelis lineis terminata nunquam aut articulata aut in margine vineta nec contenti quid in eo apparet; passim in ramulos divisa. Sporidia rotunda binis adumbrantur lineis concentricis quarum interior spatium lucidum circumdat in acervulis agminata. (Robin, op. citat., p. 436.)

To any one who will be at the pains of investigating the structure here described, it will be unnecessary to submit the arguments for its vegetable nature. The character of the branching filaments; the mode of their fructification; and the form and structure of the cells (sporules) so produced, associate it with the fungi, and distinguish it from all, even the lowest, forms of animal productions: yet there still prevails a contrary opinion.

Mr. Wilson, in discussing the vegetable nature of the epidermic growths in *Porrigo lupinosa*, makes use of the following reasoning: "From analogy," he says, "the mode of development and growth of a cell must be the same, in whatever part of the body it is produced, and whatever special purpose it may have to perform; and microscopical investigation establishes the existence of an identity of structure among them. The blood-cell, the mucus-cell, the pus-cell, the pigment-cell, the epithelial- or epidermal-cell, for example, resemble each other closely (sic) in construction, and, in some instances, appear to be convertible the one into the other. The cells or corpuscles of favus possess a striking resemblance to pus cells, and, excepting in their form, are closely allied to young epidermal cells, so that it would require no stretch of imagination to suppose the epidermal cell, altered in its actions by disease, capable of assuming the character of a pus-cell; or the latter, from a similar cause, passing into the likeness of a favus cell." After this apology for such a conversion, the author proceeds to detail his observations on the change of pus into the structures in question. The examinations which I have made of the favi in different stages, on many occasions, lead me, however, to an opposite conclusion. I could never trace any intermediate steps to support the theory offered by Mr. Wilson. The cellules, to which the name of sporules has been given, have all the physical and chemical properties of the sporules of the lower fungi, and the stages by which they develop filaments and fructify are readily traceable, which would certainly not be the case were they pus-cells, or any other form of exudation cell. An argument like this adduced by Mr. Wilson against the vegetable nature of these productions, was not many years ago used by Professor Owen against the independent animal nature of the accephalocyst



hydatid. "The knowledge," he says, "that we now possess of the primitive embryonic forms of all animals, and of all animal tissues, places us in a position to take a true view of the nature of an acephalocyst. It seems to be most truly designated as 'a gigantic organic cell,' not as a species of animal, even of the simplest kind. . . . The primitive forms of all tissues are from cells, which grow by imbibition, and which develop their like from their nucleus of hyaline. All animal tissues result from the transformations of these cells. It is to such cells that the acephalocyst bears the closest analogies in physical, chemical, and vital properties;" yet, notwithstanding this connected chain of analogical reasoning, by so acute an observer, we are at this day in a condition to see its fallacy, and to assert, on the contrary, that physically, chemically, and vitally, there is no similarity (at least such as implies identity) between the texture of the hydatid cell and of those which constitute the embryonic forms of normal textures.

Every advance in knowledge points to a similar negation of the analogical arguments adduced by Mr. Wilson, and confirms the opinion of continental pathologists, that the surfaces of the body are subject to true vegetable parasites, as they, and other textures, are to animal parasites. In any given case, therefore, it appears to be not a question of the vegetable nature of these formations, but whether they are accidental, and their presence determined by some prior morbid process, or whether they constitute by themselves a substantial and independent cause of disease. Dr. Hughes Bennett<sup>1</sup> maintains the opinion, that all such formations are secondary, and "always arise in living animals previously diseased." That a large proportion of them owe their existence to such circumstances is probable; as the *sarcina ventriculi*—the fungi found in thrush; on pulmonary tubercle; on the pleura in pneumothorax; in saccharine urine; in the sordes and mucus of the mouth; in the discharges in chronic disease of the ear; and in the decomposing evacuations in cholera. On the other hand, there are others which cannot be so disposed of, and which appear to fix on previously healthy surfaces, and to be in themselves the essential cause of disease—as the *trichophyton tonsurans*, in porrigo scutulata; the *microsporon furfur*, in pityriasis versicolor; the *microsporon*

<sup>1</sup> 'Transactions of the Royal Society of Edinburgh,' 1844, p. 291.



*Audonini*, in one of the forms of *porrigo decalvans*; and the *achorion Schönleini*, in *porrigo lupinosa*.

Though I can confirm the statement made by Bennett and Wilson as to the more frequent occurrence of this last in scrofulous subjects, yet I have seen it where the general health was unexceptionable.

In a practical point of view, the distinction between the two classes is of value, since if we have once ascertained what affections are essentially local, our treatment directs itself accordingly, instead of leaving us to hull about in a sea of conjecture as to constitutional causes and predispositions which have no existence.

As little does it avail to attempt the cure of pityriasis, and the like affections, by constitutional means, as it would of scabies. As in the latter we seek to destroy the parasitic animal and its ova, so, in the former, the fungus and its sporules. A cachexia may be present in either case, requiring appropriate treatment, but, whether present or absent, the local disease gives the same therapeutical indications. It is not the object of these notes, however, to discuss the treatment of the vegetable diseases of the skin, but I may add, that I have found the acidum aceticum diluted with four or six parts of water effectual in clearing the skin of pityriasis versicolor. The above remarks apply only to this variety of pityriasis. There are, it is well known, other forms which arise from an abnormal production of epithelium, in which no vegetable structure takes any part.

SOME OBSERVATIONS  
ON THE  
ABDOMINAL SYMPATHETIC NERVE,  
AND ON THE  
UNION OF THE PHRENIC AND PNEUMOGASTRIC NERVES.

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BY S. O. HABERSHON, M.D.

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THE sympathetic nerve has hitherto received much less attention than the nerves of the cerebro-spinal system. This is partly due to its greater complexity, as well as to its functions and diseases being further removed from the sphere of ordinary observation. It has been rightly regarded, while in a state of integrity, as of essential importance in maintaining the vegetative or nutritive functions of the body; but its diseases, as manifested in the derangements of those functions, seem to have been much overlooked.

We would here remind the reader that we distinguish between the sympathetic phenomena which are the result of a mental act and those which are purely physical in their origin, as it is to the latter alone that reference is made in this communication.

There are many facts, the explanation of which is referred to the sympathetic nerve: amongst these may be mentioned the harmony of the functions of digestion and of the circulation. By its action in health the even balance of every organ is maintained, each gland pours out its proper secretion, and those of excretory character remove noxious substances without any consciousness to the individual. Thus in digestion, the saliva is poured out in its due proportion,

the glands receiving a fuller supply of blood during their increased action. The mouth, the throat, and the whole of the tract of the alimentary canal participate in the act, while the gastric, the pancreatic juices and the bile are regulated in time, and in quantity, for the proper furtherance and completion of the function. Thus all the actions of these parts are suited the one to the other. Such a harmony as this, as well as that of the circulation throughout the whole system, is due to the operation of the sympathetic nerve, an organ which appears in fact to preside over the whole chemico-vital functions of the body.

It is, however, in disease, and in the derangements of this harmony, that we are led now more attentively to consider its action. Thus the gastric juice may be secreted when it is not needed, or may be deficient in quantity during digestion. This sympathetic action is seen when a fright or great nervous excitement removes craving hunger, or interferes with digestion, or leads to flatulent distension. It is seen by the sinking sensation of exhaustion and emptiness in the abdomen in dyspepsia; by the violent vomiting in disease of the uterus or the kidneys, where the stomach itself may be unaffected; and again, the sympathy with the organs of circulation is seen by the failing pulse in severe abdominal disease, by the irregular intermittent pulse in dyspepsia, as well as in many affections of the brain, as depressions of mind, disordered sensations, hypochondriacal feelings arising from gastric or intestinal disturbances. All these have their origin in the connection which the sympathetic has with the several organs and functions of the body.

The cerebro-spinal nerves are, as is well known, brought into close relation with the sympathetic; but whether, according to the theory of Bichat, the two systems are in themselves distinct, or whether they form only one, according to the assumption of Valentin, is doubtful. Many arguments may be adduced on either side.

As to the arrangement of the sympathetic, a series of ganglia are placed in tolerably close proximity to the whole of the spinal nerves, as they emerge from the vertebral canal; other ganglia and plexuses are connected with the heart, with the digestive, and urino-genital organs, with the salivary and



lachrymal glands, &c.; wherever they exist, however, their elements bear a close resemblance to one another. They are composed of large nucleated cells, the nucleus and nucleolus of which are very distinct, the cells themselves being surrounded by smaller nuclei, often in several layers, and so constant are these in their character, in man, that they are doubtless important elements in the ganglia.<sup>1</sup> Some anatomists have considered these nuclei as a form of fibrous tissue; but of this there is no satisfactory proof, though supported by the authority of Bidder, Volkman, Kölliker, &c. Extending from the cells, sometimes from opposite extremities, when bipolar, or from several parts, there are minute processes which become at length delicate fibres, although some cells appear destitute of them. Between the cells are other fibres resembling the ordinary spinal nerves passing into or through the ganglion. The delicate fibres are often found to be exceedingly small, and are by some considered as the true sympathetic nerve fibre. A single double-outline nerve fibre may often be observed, having elongated nuclei placed on either side. On the one side of the ganglion, then, we find the ordinary spinal nerve proceeding to it, and upon it delicate or nucleated fibres, and the same also obtains on the opposed side: the relative proportion of these forms of nerve fibre varying exceedingly.

This arrangement of parts is evidently well suited to bring the spinal nerve into relation with the ganglionic cells, if such be centres of force; for although we have no proof of anything allied to the inductive action of electro-galvanism, or that the nerve force is identical with electricity (the sympathetic fibre, moreover, being destitute of the insulating medium of the spinal nerve), still the arrangement certainly would suggest such an idea; and it may be that there is the same difference, or an allied one, between the forms of nerve force as between electricity and galvanism. However this may be, the connection is a very intimate one, and is shown in the pain which arises from disturbed functions of several organs. Thus the pain which we observe in the shoulder in hepatic disease arises

<sup>1</sup> For the minute anatomy of these ganglia, their structure, and development, I must refer to the valuable article by Dr. Drummond, in 'Encyclopædia of Anatomy and Physiology;' to Todd and Bowman's 'Physiology;' to Müller's 'Physiology;' Kölliker's 'Mikroskopische Anatomie,' &c.



from the connection of the phrenic with the hepatic nerves, and the former with the spinal sources of origin of the descending branches of the cervical plexus. Again, the neuralgic pain in the side, in gastric and uterine disease, is from the connection of the splanchnic nerves at the spine with the fifth and sixth dorsal nerves, &c. So also the pain in the inner side of the arm arises from the close connection easily traceable between the cardiac nerves and the inferior cervical ganglion, which sends a large branch to the ulnar nerve. Many other instances of this kind might be mentioned.

The ganglia to which we have referred have been considered by some physiologists, as by Henle, by Volkman, and by Grainger, &c., as themselves centres of reflex action; by Müller, Valentin, and others, it has been thought that the reflex actions, apparently dependent on them, are really produced by the spinal system. I am disposed to agree with the former, and with the views originally expressed by Remak, who considered the manifestations of reflex action of the sympathetic to be of different kind from that of the cerebro-spinal system.

Many experiments have been made to determine the function of the sympathetic, and some of these have been productive of no good, whilst others appear worse than useless, for instead of adding any new fact they have tended rather to misguide. In many, the shock of the operation itself would invalidate the whole experiment. What reliance, for instance, can be placed upon a *vivi-section* by which the abdominal cavity is opened to determine the function of the sympathetic? Suppose the instance of a human being suffering from hernia or a perforating wound of the abdomen: he would certainly not be in a favorable condition to determine the healthy performance of digestion. The attempt has been made to test the sympathetic by the observance of motion or sensation, but the occurrence of which phenomena would perhaps only be due to its connection with the cerebro-spinal system, and not dependent upon any part of its own function. The contraction of the pupil by irritation of the sympathetic nerve in the neck is an instance of this kind, being produced probably by its connection with the third nerve, rather than by its own direct action.

The experiments of Bernard in reference to increase of temperature in the neck, by division of the cervical sympathetic, are of great interest, in whatever way explained. The function of the sympathetic is shown in the modification of secretions, in disordered functions, and in altered temperature, rather than by pain, which, if it do occur, is to be attributed to its connection with the spinal nerve. Careful examinations after death would often reveal cases in which branches of the sympathetic are destroyed; which facts, if previously associated with careful observations as to the symptoms during life, might constitute most valuable indications of healthy function. Such dissections, however, require considerable time; and great care is called for in estimating whether the symptoms noticed during life had any other origin. In cancerous diseases, in aneurism, in inflammatory or other growths, we find instances in which these nerves are sometimes entirely destroyed.

It is to one or two portions of the abdominal sympathetic that I wish to draw particular attention.

The semi-lunar ganglion is situated in front of the vertebræ close to the origin of the celiac axis, and extends to both sides of the aorta. It is composed of two portions or sets of ganglionic masses, a right and a left, which are united by large branches above and below the celiac axis and the superior mesenteric arteries, constituting the solar plexus. On the upper or diaphragmatic side, the ganglia receive the splanchnic nerves, by which they communicate with the dorsal spinal nerves. The pneumogastric joins it about the median line, or towards the right ganglion, by a large trunk formed by branches from both right and left pneumogastric nerves, and on the left side a much smaller branch passes directly to the semi-lunar. On tracing the phrenic on both sides it is found to send a branch onwards to join one extending upwards from the semilunar ganglion, and these branches join with others sent from the pneumogastric near the base of the pericardium. These branches form, on the left side, a delicate plexus between the phrenic, pneumogastric, and sympathetic; but, on the right, a more direct branch is found passing from the pneumogastric before it reaches the semilunar ganglion, and extends behind the lobulus spigelii, close to the vena cava, to join a branch from the phrenic; a large branch is also given off from

the semilunar ganglion, and forms the diaphragmatic ganglion. This is a triangular ganglionic mass receiving the three nerves just mentioned, and giving off several branches to the diaphragm, and two long branches extend to the anterior surface of the lobulus spigellii. The union of these nerves is found to exist on both sides; but is more intimate on the right, and brings into association the two most important nerves of respiration, the phrenic and pneumogastric, with the semilunar ganglion, the great centre of the nerves of digestion and of the alimentary canal. Branches are given off from the large semilunar ganglionic masses to the several viscera of the abdomen, upon the hepatic artery to the liver, upon the coronary to the stomach, to the spleen, to the intestines, to the kidneys, &c., as well as upon their arteries, and smaller branches extend upon the aorta.

The suprarenal capsules are situated close to the semilunar ganglia, and receive numerous large branches. On the left side a filament may easily be traced directly from the pneumogastric nerve, and on the right, several branches extend from the nerve upon the diaphragmatic artery. Many of these extend upon the surface of the capsule, others penetrate into the interior: some of these larger branches I have dissected into the suprarenal capsule, and have found them forming loops, and again leaving the capsule, or having an enlargement in the capsule itself, and afterwards leaving near to the point of entrance; this may perhaps be a constant arrangement, but the connection is evidently a very intimate one between the suprarenal capsules and the large sympathetic nerves of the abdomen, and also with the pneumogastric.

These branches, to which reference has been made, may be seen on the drawing made from my dissection of these parts. (See Plate VI and VII, fig. 3.)

The union of the phrenic, pneumogastric, and splanchnic nerves, is a very interesting one. The use of the diaphragmatic ganglion appears to be to bring the diaphragm into intimate relation in its action with the abdominal viscera, and to unite the digestive and respiratory and cardiac centres of the sympathetic nerve. The pneumogastric and phrenic are the most important nerves of respiration; and here they are



brought into close connection with the splanchnic, the great nerve of digestion, and send branches to the liver, the largest of the abdominal glands.

How closely does the diaphragm sympathise in its action with diseases of the abdomen ! In peritonitis the respiration is performed by the muscles of the thorax, to allow rest to the inflamed membrane ; in vomiting, in disease of the stomach, or of the uterus, or of the kidney, the diaphragm is probably brought into violent action. Again, observe the effect of disease of the abdominal viscera on the heart as well as the whole circulation. A blow on the epigastrium has often been followed by sudden death ; in many cases of peritonitis, in gastritis, or ruptured intestine, or in hernia, observe the collapse, and the shock which the whole system has sustained, the coldness of skin, the compressibility of the pulse, the anxious countenance, the abdominal distress, and speedy death, although the mind is unaffected, and sensible to the last ; and this is not due to the operation of the cerebro-spinal, but of the sympathetic nerve. Not less marked is this connection in chronic abdominal diseases, and even in affections of the stomach of a functional or temporary character, as every dyspeptic can testify, by the throbbing throughout the whole frame, or the conscious pulsation of the entire vascular system during the severity of his attacks. The pain in the shoulder which is sometimes observed in disease of the liver, arises probably from this connection of the phrenic with the splanchnic nerves by means of the diaphragmatic ganglion. The phrenic nerve arises in the neck, from the third, fourth, and fifth spinal nerves, and the descending branches of the cervical plexus, which are distributed over the shoulder, receive their spinal origin at that part ; but this pain is *not* constant in disease of the liver ; in several cases where large nerves, forming the hepatic plexus, have been injured by disease, no pain has been observed, and possibly for this reason, that the branches which extend to the liver, along the hepatic artery, are removed from this ganglion, not brought under its relationship, and thus the cervical, spinal, and phrenic nerves are not affected. The diaphragmatic ganglion is separate from Glisson's capsule, and is situated on the cava.

In the same manner, pain down the arm is not *constant* in disease of the heart, for serious valvular disease occurs without



it; but in aneurismal disease of the aorta, &c., where the large cardiac nerves become pressed upon, the connection with the ulnar nerve is shown by the pain along the arm.

The diseased condition of the suprarenal capsule, as shown by Dr. Addison, and the symptoms consequent on that disease, point strongly to the sympathetic.

Dr. Gull has expressed the opinion, that the suprarenal capsules are allied in structure to the pituitary body in the brain; whether such be the case or not, we find a very close union with the semilunar ganglion, and with the pneumogastric nerves. Dr. Beck shows these branches in his dissections of the sympathetic, published by the Royal Society.

In the symptoms of renal capsular disease, these relations are brought out in a very marked manner; the loss of appetite and of strength, the irritability of the stomach, the sense of exhaustion, the failing pulse, and the gradually sinking powers of life, arise from the sympathetic nerve being involved, and not the cerebro-spinal.

The examination of the semilunar ganglion, in cases taken from the general post-mortem inspections of the hospital, show that they are subject to some pathological conditions easily recognisable; the colour varies much, sometimes being pale, at other times quite of a deep chocolate. This latter change arises from the deposition of a great number of grains of pigment in the large ganglionic cells. More or less of this pigment always exists; but in the following case the large nuclei and nucleoli were almost obscured by it. It was that of a man sixty-five years of age, who died from cancerous disease of the glands of the axilla and neck, of the pleura, and of the lumbar glands, no symptoms were observed which could be attributed directly to this condition; he became increasingly prostrate, and died from exhaustion. It is probable that this state was a form of pigmental degeneration, possibly merely connected with the age and the enfeebled condition of the patient, but the obliteration of the large branches of the sympathetic nerve certainly do appear sometimes to be followed by wasting and diminished nutritive power in the organ from which the supply has been cut off.

I have traced with considerable care the hepatic branches of the semilunar ganglion, in cases of organic disease of the liver;

these, in many instances of cirrhosis, where a great quantity of fibrous tissue had been poured out at Glisson's capsule, were observed to pass free from apparent injury, and extended along with the larger branches of the portal canals; in others, they appeared involved in the tissue, surrounded, but could be traced throughout it. In two cases, the slow development of cancerous tumours in the immediate neighbourhood of the vena porta, led to the complete destruction of the large sympathetic nerve extending into the gland. In both, the liver was exceedingly wasted, and the observation would have been of great pathological interest, had it not happened that there was another cause for this atrophy, namely, obstruction of the common duct, and considerable distension of its branches throughout the whole of the liver; the destruction of these nerves, then, could not be assigned as the cause of this loss of function. The most distressing symptom which one of these patients suffered from, beyond the sense of weakness and exhaustion, was diarrhœa, for which there was no ulceration or apparent disease of the mucous membrane to account. The other suffered severely from pyrosis and heartburn; there was slight pressure of the duodenum, but not sufficient apparently to cause obstruction to the passage of the chyme; no vomiting, but merely this most distressing heartburn. It may be that these symptoms, the diarrhœa in one, the heartburn and pyrosis in the other, arose from causes which are not apparent, the latter, perhaps, from retained gastric secretions. It is certain that they are evidence of disordered secretions in the mucous membrane of the stomach and alimentary canal, and that in these cases there was serious implication of large trunks of the sympathetic nerve; but whether these disordered functions arose from irritation of the sympathetic nerve may be problematical. The observation is merely recorded, and the careful dissection and notice of allied cases might reveal to us definite knowledge in reference to some obscure points both of physiology and pathology. These are experiments made for our observation by disease, and are of infinitely more value than applying ligatures to nerves deeply seated, or dividing these nerves during life, or the applying electro-galvanic currents, all of them uncertain, fallacious, and exceedingly obscure in their result.

In Case iv, recorded, with cancerous disease of the œsophagus, in my communication on dysphagia (the following article in this volume,) the right semilunar ganglion itself appeared to be infiltrated with cancer: the abdominal viscera and glands were wasted, but the original disease was so extensive, and of so long duration, the period of life so advanced, and exhaustion so extreme, that it is doubtful how far the symptoms could be attributed to the injured ganglion.

# ON DYSPHAGIA.

ILLUSTRATED BY

## SOME CASES OF DISEASE OF THE ŒSOPHAGUS AND PHARYNX.

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BY S. O. HABERSHON, M.D.

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THE Œsophagus is a portion of the alimentary tract more than some others exempt from organic disease, and for these reasons: in the first place, its function is of an exceedingly simple character, being merely to conduct the food into the stomach; and, secondly, the transit of the food is very rapid over its mucous membrane. The close contact of the Œsophagus with many important structures at the root of the lungs sometimes leads to its becoming involved in disease originating in those parts, although this is not so frequent as primary disease of the Œsophagus implicating the latter structures.

The pharynx is the organ of deglutition, and disease of any part of it, or of the openings into it, leads to difficulty in the performance of its function, or dysphagia; but the process of swallowing can scarcely be said to be fully completed until the food is lodged in the stomach; and hence dysphagia becomes one of the most prominent symptoms of disease, not only of the pharynx, but also of the Œsophagus.

The causes of dysphagia are very varied, and some are well illustrated in the cases of the present communication, which have occurred at Guy's Hospital. They are chiefly as follows:

I. From disease of the tonsils or palate.

II. From inflammation of the cellular tissue of the pharynx or Œsophagus.



III. From disease of the laryngeal cartilages, or epiglottis.

IV. From functional or spasmodic stricture of the œsophagus or pharynx, as in hysteria, hydrophobia, &c.

V. From paralysis of the muscles.

VI. From acute inflammation of the mucous membrane.

VII. From mechanical injury or poisons.

VIII. From structural obstruction to the œsophagus, as—

1. Constriction ;
2. Ulcerations, sometimes communicating with the larynx ;
3. Cancerous disease ;
4. Obstruction from the pressure of aneurismal or other tumours.

I. *Dysphagia arising from cynanche tonsillaris*, as well as from acute inflammation of the throat, from scarlet fever, from diphtheritic inflammation, &c., is of varied frequency. Cases of it, arising from some of these causes, are so often observed that they do not need special mention here.

II. *Inflammation of the cellular tissue of the neck*, associated with pyæmia or with erysipelas. In my notes I find the following case, a very interesting one of the kind, occurring in 1847 :

Abraham Stanley, æt. 36, a sailor, of intemperate habits, was admitted into Guy's on October 13th, 1847. On the 5th, whilst unloading coals, he received a blow on the back of the hand, and on the following day rigors came on, and pain in the axilla, but the skin of the arm did not become inflamed. On admission, on the 13th, he presented the appearance of a man suffering from typhoid fever ; there was delirium at night ; pulse very soft, 106 ; the tongue moist, and the respiration much oppressed ; no fluctuation could be found under the pectoral muscle, or any suppuration detected in the neck ; and the wound on the hand was dried. Stimulants and opium were administered on the 15th. The respiration was difficult and laboured, 42 per minute ; there was evident obstruction of the larynx, and there was some tenderness about it, but scarcely any swelling, and no fluctuation or suppuration could be detected on very careful examination ; there was also great difficulty in swallowing. On the 16th, the respiration and deglutition were somewhat easier, but the skin was clammy, and the tongue dry. He died on the following day, after

vomiting some blood. On inspection, the whole of the cellular tissue surrounding the muscles of the neck were found infiltrated with pus, but there was none below the pectoral muscle.

In another case, which was admitted in May, 1847, of a woman, aged 66, there was sore throat, with pyrexia, quickly followed by typhoid symptoms, and death on the fifth day. On inspection, suppuration was found among the muscles of the neck, which extended round the œsophagus as low as the root of the lung. In the pharynx there were several superficial ulcers, and one opposite the arytenoid cartilage had extended into the cellular tissue. The disease appeared to be an erysipelatous form of inflammation, and of such an aggravated kind as to be quite beyond the reach of remedial measures.

III. *Disease of the laryngeal cartilages, or epiglottis.*—Disease of the thyroid or cricoid cartilages rarely, except in cancer, extends to the pharynx, but much more frequently leads, as in necrosis of these parts, to suppuration among the muscles of the neck, or to chronic laryngitis of a most intractable form. We seldom see great difficulty in swallowing from disease of these cartilages, but the reverse is the case when the epiglottis is affected, whether the ulceration arise from syphilitic, phthisical, or cancerous disease. In syphilis both the glossal and laryngeal surfaces of the epiglottis become diseased, and sometimes nearly the whole is destroyed, leading to distressing dysphagia; and in phthisis this ulceration of the epiglottis is one of the most trying complications of the complaint, the ulceration extending on its inner surface as far as the margin, which becomes eroded and gradually destroyed, so that the contact of food, &c., with this irritated surface, leads to its instant rejection, sometimes through the nares. In chronic phthisis I have seen this condition attributed to organic disease of the œsophagus itself, from the extreme urgency of the dysphagia, and from the food appearing to have passed below the pharynx before it was forcibly ejected.

IV. *Spasmodic stricture of the œsophagus.*—The few cases of this kind which have come under my own observation have been in young women of an excitable nervous system, with

leucorrhœa or painful menstruation, and impaired digestion. The strongest language was used by these patients to express their inability to swallow, and they showed the greatest unwillingness even to attempt it. One of these was a young woman about 23 years of age, thin, and imperfectly nourished. No obstruction whatever was found on passing an œsophageal bougie, and she afterwards swallowed food in small quantities, increased day by day until she took the usual amount. Lesser degrees of this condition are not unfrequent in *hysterical* subjects; and, as the symptoms of hysteria are well marked in them, there is little danger of mistaking the complaint for cancerous obstruction, although there may be greater difficulty in diagnosing it from perforating ulcer extending into the trachea.

The most marked true spasm of the pharynx and œsophagus is found in *hydrophobia*. Two years ago a case of this terrible disease occurred at Guy's. On post-mortem inspection, besides great congestion of the membranes of the brain and spinal cord, the pharynx was the only part affected, and the appearance here was very peculiar. The organ appeared more than twice its natural capacity; the constrictor muscles retracted to the utmost; the fauces exceedingly large, from the rigid contraction of the soft palate; and every part appeared expanded to the utmost. The mucous membrane was injected, and covered with some mucus. The œsophagus, also, was contracted; the lungs intensely congested; the other viscera healthy; but there was emphysema of the neck. The symptoms during life indicated extreme irritability of the nerves supplying the pharynx—in fact, all the branches of the fifth and pneumogastric nerves.

The patient was a young man, aged 23, who was said to have been bitten by a dog nine years previously. On the day of admission into Guy's, May 15th, 1854, difficulty of swallowing came on, and great mental excitement. He was removed to one of the adjoining workhouses, and afterwards brought to the hospital, about nine o'clock in the evening. He was a strong muscular man, and at first sight appeared to be affected with acute mania, or delirium tremens; but there was a sudden starting, especially when a draught of cold air came in contact with his face, which more clearly indicated the character of the disease. This starting evidently resulted from spasmodic action of the muscles of the face and pharynx; his countenance had a wild and excited aspect; he thought that he was being murdered, that boiling water was dropping upon his face, and he said that he felt choked. The pulse at nine



o'clock was 90, at eleven, it was 120; the tongue clean, the pupils widely dilated, the face bathed in sweat, the hands clammy; he would not attempt to drink, but dashed the cup away from him with a violent spasmodic action, but he ate a small portion of bread; he was frequently spitting out saliva. Restraint was required, for in his terror, which was fearful to witness, he rushed at the window, and would have seriously injured himself. I remained with Dr. Gull for several hours during the night with this patient, a witness of one of the most fearful spectacles of misery and disease I have ever seen. About 12.30, an injection, containing 10 grains of cannabis indica, was administered; the whole of the enema was at once returned. At one o'clock a longer tube was passed, and the same quantity again injected; the paroxysms had then become very violent and frequent, and the pulse exceedingly small, varying, and occasionally intermittent, 120 to 130 per minute. At 2.15, he was still more violent, calling out as loudly as his strength would permit him. It was then determined to administer chloroform. Intense congestion of the eyes and face came on; the pupils became much smaller, the pulse a little more perceptible; the respiration, which had been catching and accompanied with gasping and sighing, became more regular. In four or five minutes after leaving off the chloroform the paroxysms began to return; the face, however, did not at once become sensible to impression. Chloroform was administered three times during the hour, and on leaving it off the same return of paroxysm took place; the pulse became almost imperceptible, and the respiration more stertorous. About 3 a.m., whilst under the influence of chloroform, 10 grains of cannabis indica were placed in his mouth; it became mixed with saliva, but was all ejected. He died at 3.30, from exhaustion and apnœa.

V. *Paralysis of the muscles of deglutition* is generally observed immediately to precede those of respiration; and is looked upon, correctly, as a common sign of approaching death. The nervous centre of the function of swallowing is close to that of respiration, and there is an intimate connection between them. Where there is this loss of power, placing fluid in the mouth will be followed by its entrance into the larynx, or by violent cough, or it may even hasten death.

We not unfrequently, however, observe, in cases of hemiplegia, where the muscles of the tongue are paralysed, that swallowing becomes exceedingly difficult, especially with solids. This difficulty appears to arise from the movements of the tongue being restrained, the bolus of food cannot be formed, and pushed back into the fauces; fluids are more easily swallowed, because more readily brought to the action of the true muscles of deglutition.

Another class of cases are those connected with mental disease, some of which may easily be mistaken for true paralysis. With great feebleness of muscular power, we may find that the will is unable to excite muscular action; that the mus-



cles of the pharynx appear paralysed, because they are not stimulated to healthy contraction, and hence deglutition becomes apparently impossible. The following interesting case, admitted under my care, into Job Ward, July, 1856, is of this kind.

He was an emaciated man, aged 60, a gas-fitter, residing at Deptford, of a dingy, sallow appearance. His wife stated, that for several years he had been occasionally irritable, and that his only complaint was of pain in the region of the transverse colon. On the 18th, he appeared to lose the power both of speaking and swallowing, having previously said, that "he did not know what was coming over him." On the 23d, he was brought to Guy's; he appeared prostrate, unable to stand, but could slowly move his legs and his arms; his countenance was not without intelligence, and he appeared slightly to understand questions; he could not protrude his tongue, which remained almost motionless at the floor of the mouth, and dry on its surface; fluids put into the mouth, were retained, and ran out again at the margins, and he could not be induced to attempt to swallow; placing a teaspoon at the back of the mouth excited some action of the muscles; the pupils were active, the right rather larger than the left; the pulse 56, and compressible; the heart's action very feeble; respiration normal, 20 per minute, but the air could scarcely be heard to enter into the chest. The abdominal muscles were exceedingly rigid, but the abdomen not distended. Half a drop of croton oil was placed on the back of the tongue, and afterwards, a nutrient enema was administered. On the 24th, my colleague, Mr. Cooper Forster, passed an œsophageal tube into the stomach without any difficulty, or meeting with any obstruction; some beef tea thickened with arrow-root was in this way administered; the patient afterwards swallowed milk and beef tea, &c., with less and less difficulty, and on the third day began to speak; the bowels were acted on by castor oil, and by enemata; ammonia and calumba, with a small quantity of wine, were given on account of his prostrate condition; his tongue lost its brown and furred condition, and he rapidly improved. His mind, however, was not in a clear state, for as soon as he was able to eat, he had the idea that no other patient in the ward had any food. This case closely resembles those found in asylums, where the patients refuse to eat; but here, there appeared to be the inability to make the attempt to swallow—a condition which might easily have been mistaken for paralysis of the muscles themselves.

VI. *Inflammation of the mucous membrane.*—I have not seen any instance in which the ordinary indications of inflammatory action were observed in the œsophagus after death; but this may be due to their disappearance after life has ceased.

In a case of acute inflammation of both small and large intestines of a diphtheritic character, in a woman æt. 28, admitted under my care, into Guy's Hospital, during last year, the mouth was inflamed, and the pharynx and tonsils were covered with a white film, spread upon an injected mucous

membrane. This white film consisted of a beautiful torula, interlacing in all directions, constituting the *muguet*; it extended downwards to the commencement of the œsophagus, and some traces of it were found in that canal. In this instance, the symptoms were those of dysenteric diarrhœa, which had continued for several months before her admission into Guy's, and had persisted without any intermission for seven weeks. The disease was attributed to her removal into a damp house. The patient was exceedingly prostrate, and with the diarrhœa had distressing vomiting, retching on attempting to take food, which also produced severe pain. No medicines or injections had any effect in checking the diarrhœa, and she died on the third day after admission. It appeared, indeed, that the whole tract of the alimentary canal, from the mouth to the rectum, was inflamed; nearly the whole of the mucous membrane of the colon was covered with a whitish adherent layer, on a thickened membrane, presenting several aphthous ulcers; the ileum was intensely injected; the liver fatty, and lighter than water; the lungs, pleura, heart, &c., quite healthy.

It is probable, that in some of the cases of severe gastro-enteritis in children, in whom the mouth as well as the intestine is evidently inflamed, the whole of the alimentary tract is affected, and would present before death a condition quite abnormal. At the close of chronic disease, we find a similar condition of the pharynx, rendering deglutition both painful and difficult; aphthous inflammation of the mouth having extended into this part. In these conditions, I have not seen any remedy followed by such beneficial effects as the chlorate of potash, associated sometimes with borax and honey; but alone it often acts apparently in a most marked manner. This remedy in stomatitis was very extensively used by the late Dr. Golding Bird, and subsequent observers have confirmed the opinion which he entertained. It appears to act partly by its local effect, and also as a saline after its absorption into the system.

VII. *Dysphagia from destruction of the mucous membrane by mechanical or chemical agents.*—Many instances occur year by year of infants drinking boiling water; in these cases the

vesication produced is seen in the pharynx and on the epiglottis, and often in inflammation of the larynx and trachea rather than of the œsophagus, the water being violently ejected before much of it can be swallowed. In several of the inspections after death, it has been found that the lower part of the œsophagus and stomach presented considerable congestion, showing apparently that some of the hot water had reached those parts. With corrosive poisons the effects must be divided into those which are immediate or primary, and those which are remote or secondary. Generally the mucous membrane becomes charred and destroyed by the direct chemical action of the poison, as from sulphuric acid, and death takes place in a very short time ; but, if the patient recover from the first effect, and the mucous membrane of the œsophagus be destroyed, an inflammatory product is effused into the sub-mucous cellular tissue, thickening and contraction take place, and in this manner, an annular constriction of the œsophagus may arise.

In an interesting case of poisoning by sulphuric acid, in October, 1855, in which death did not take place until the eleventh day, the mouth and throat were of a whitish colour ; at the posterior part of the mouth, there was considerable injection of the mucous membrane, and on each side of the posterior pillar of the fauces there were whitish loose patches of membrane. The edge of the epiglottis was found minutely eroded, and the mucous membrane of the œsophagus was pale and covered with yellow membranous flakes. In this case, the prostration and collapse immediately following the reception of the poison were accompanied by vomiting of grumous blood, but in less than twelve hours the patient was able to swallow some milk and arrow-root ; and on the fourth day appeared to take her food without difficulty. Death took place from the sloughing condition of the mucous membrane of the stomach, combined with the inflammation of the duodenum, and in fact with that of the whole tract of the intestine. The ability to swallow in this case is seen to have been restored in a very short time, considering the fearful injuries which resulted to the whole of the mucous membrane.

The following case illustrates the *secondary* effect of a corrosive poison in the thickening of the whole of the œsophagus and obstructed pylorus, which led to a fatal termination, in a man who died three months after having taken one ounce of nitric acid.

James T—, æt. 24, was admitted under Dr. Barlow's care, in March, 1852, in a state of extreme emaciation ; he vomited, with some pain, all the food which he had



swallowed; the abdomen sometimes became extremely distended; the bowels had only been opened twice during the two months preceding his admission; the tongue was injected. He lived eighteen days after admission; but his symptoms gradually increased from the time of taking the acid, which he had done by mistake, not discovering the accident until he had completely swallowed it. On inspection, the epiglottis appeared healthy; the mucous membrane of the whole of the œsophagus was thickened and readily separated; the submucous tissue and all the coats of the œsophagus were also thickened; the stomach was enormously distended, reaching to the anterior superior spinous process of the pubes; the pylorus was obstructed, thickened, and contracted; the lungs and heart were healthy; the liver was small, deep in colour, and the gall-bladder contained about  $\frac{3}{4}$ ss of dark-coloured bile; no other viscus was diseased.

VIII. *Organic obstruction of the œsophagus.*—Cases of this kind may be divided into those arising from—1, annular constriction; 2, ulceration, sometimes communicating with the trachea; 3, cancer; and 4, pressure from aneurismal or other tumours. The history and close attention to the symptoms will alone enable us to distinguish these cases from one another. In some of these it is to be hoped, that remedial means may be used which have hitherto not been attempted; in others, it is evident that nothing can be done for cure, but at the same time pain may often be obviated, and some of the symptoms mitigated.

*Annular constriction of the œsophagus* consists in the effusion of fibrinous material into the submucous cellular tissue; this tissue contracts, and becomes exceedingly dense, forming a firm constricting band, while the tube above dilates, and the obstruction increasing, at last the passage of food becomes impossible. A beautiful specimen of this form of constriction is shown in the Preparation No. 1789, in the Museum of Guy's. Many of these cases are probably the result of corrosive poisons, or arise from injury to the mucous membrane, or from inflammation set up by adjoining disease. No. 1789<sup>50</sup>, in the Guy's Museum, shows the œsophagus of a lad, in which about two inches above the diaphragm the parietes of this tube are thickened, the mucous membrane contracted, and apparently cicatrized for the space of an inch and a half. There is an absorbent gland in the neighbourhood of the stricture, adherent to the walls of the canal. The boy had had difficulty of swallowing from infancy, and a bougie had



been passed occasionally. Inflammation about the gland had, perhaps, led to this thickening of the canal.

An exceedingly interesting case, bearing some relation to the preparation just mentioned, was recorded by Dr. Ogier Ward, in the 'Transactions of the Path. Soc.,' for the year 1850. A boy, ten years of age, who died from meningitis, suffered from difficulty of deglutition a month before his death, which disappeared, however, before his last illness. On inspection, a longitudinal ulcer was found at the bifurcation of the trachea, communicating with a suppurating bronchial gland.

In recorded cases of annular stricture, the obstruction has gradually increased in severity, and unless we had a history of poison having been taken, or the discharge of pus from abscess, I know of no direct symptom by which they can be distinguished from those of so-called cancerous disease. The passage of a bougie may reveal to us the presence of obstruction, without indicating its true character, unless mucus from the bougie present us with cancerous products.

*Ulceration.*—In the Museum at Guy's, there are several specimens showing ulceration of the œsophagus, of a non-cancerous character, extending into the trachea, and there is some obscurity as to the correct pathology of them; difficulty of deglutition was the most prominent symptom during life; in some this had been gradually, in others, deglutition had suddenly become impossible; the pain was situated at the sternum or between the shoulders, and attempts at swallowing were followed by urgent dyspnœa, and the food was forcibly ejected through the nares. The patients became emaciated, and life was prolonged for a short time by the use of nutrient enemata. On inspection after death, the only disease found has been perforation of the œsophagus opening into the trachea; the openings extending over one or two inches, and two or three in number, the edges smooth, without any thickening, and in several cases the opening into the trachea being the smaller. The examination of these cases does not give any evidence of cancerous disease; the early symptoms appear to arise from the œsophagus, the difficulty in respiration following that of deglutition; nor do we find other evidence of disease either in the larynx or lungs. These facts

appear to show that the disease has not commenced either in the mucous membrane of the trachea or in disease of its cartilages, and we are led to suppose, either that an abscess has formed between the œsophagus and trachea, and led to fistulous openings into those canals, or that ulceration has taken place in the œsophagus, and gradually extended in depth through the adjoining structures. It sometimes, however, happens, that ulceration extending into the œsophagus arises from disease of the tracheal cartilages, and the following remarkable specimen is of that kind.

The case was that of a carrier, aged 42, at Hampton, who was under the care of Mr. Holleston and Mr. Jepson, in 1853. He had had crowing respiration, abundant expectoration, but no very urgent dyspnœa, or difficulty in swallowing. He gradually sank. Six months before his death, he expectorated a portion of ossified tracheal cartilage (Preparation 1711<sup>87</sup>), and six weeks later a second portion. On inspection, at the commencement of the œsophagus, immediately beneath the cricoid cartilage, was a vertical opening, half an inch in length, extending into the trachea, the edges smooth and rounded; there were three other communications, resembling fissures, being merely separated by shreds of mucous membrane. (See Preparation 1711<sup>87</sup>.) The cartilages of the trachea were ossified, and there was some ulceration of the mucous membrane of the larynx at the cricoid cartilage. The inferior lobe of the right lung was consolidated, but no other part of the body was diseased; and there was no trace of cancerous or strumous disease. Dysphagia was almost absent, as far as can be learned, in this case; and the symptoms were those indicative of disease commencing in the larynx; thus differing remarkably from the cases presently to be recorded, where dysphagia was the most prominent complaint of the patient. It is probable that their pathology is also different. No history of syphilis is given, but the expectoration of a portion of diseased cartilage, six months before death, indicated the character of the disease.

One of the cases of ulceration of the œsophagus, a woman, was admitted, several years ago, into Guy's, under Mr. Key's care, and believed to have stricture of that part. (Preparation 1714). Another occurred last year, under the care of

Dr. Barlow, and is fully reported by Dr. Wilks, in the sixth volume of the 'Pathological Transactions.'

A married woman, æt. 24, who had never had robust health, about a year previous to her admission, had enlarged glands about the neck, which diminished under the use of iodine; and six months afterwards she began to experience difficulty in swallowing, pain in the chest, uneasiness in the throat, and some shortness of breath. These symptoms increased in severity till admission, but a short time previously, had suddenly become very much aggravated. She was much emaciated; no swelling could be found about the neck, or disease of the chest. She experienced the greatest difficulty in swallowing fluids, and food was at once forcibly ejected. Mr. Hilton passed an œsophageal tube, and found that when the patient breathed, air passed from it, indicating a communication with the trachea. She was fed for six weeks entirely by injections. On inspection, the trachea and œsophagus were found extensively diseased from the cricoid cartilage, nearly as far as the bifurcation of the former, and the two communicated by three openings. The anterior wall of the œsophagus was destroyed, with the exception of two slips of muscle, which still remained; and at this part there was an oval ulcerated opening passing into the trachea; below this was a small portion of the calibre of the œsophagus remaining, but considerably contracted; below this the œsophagus was again wanting, and two more openings passed into the trachea. At this part, the posterior wall of the œsophagus was also destroyed, and the body of the last cervical vertebra exposed. The cellular tissue in front of the trachea, with the remains of the œsophagus, and the muscles of the neck, bounded the space involved by the ulceration; the openings into the trachea were oval, transverse, and perfectly smooth; not the least thickening or heterologous deposit could be detected by careful examination, aided by the microscope; the surfaces were covered with mucus. In the ovary, and in an adhesion on the surface of the liver, were slight strumous granular deposits; but no other viscus was diseased, nor was there any evidence of cancerous disease. There was slight difficulty of breathing, and Mr. Hilton performed tracheotomy, but without any permanent advantage to the patient. (See Preparation 1714<sup>10</sup>, and drawing 246<sup>24</sup>.)

The following case occurred in Guy's, in the year 1840. There is no history of the symptoms on record; but the patient was a man aged 33 years, and he died four days after admission. The post-mortem inspection was as follows:

The body was exceedingly emaciated. Near the middle of the œsophagus, the mucous membrane, for about two inches, was of a very red colour, and irregular from ulceration; the canal was much contracted, and would have scarcely admitted the end of the little finger. Below the stricture, the œsophagus was much dilated, and an abscess had formed behind it, containing four ounces of dark fluid of a sour odour; there was a small sinus leading to the abscess; the mucous membrane, both above and below the diseased part, was quite healthy; there was no evidence of cancer in the affected part; nor was any other organ diseased, except that the



kidneys were found to be granular. (See Preparation 1789<sup>75</sup>.) It was supposed, that a corrosive poison must have been taken, but of this there was no proof or evidence.

An exceedingly interesting case will be found recorded by Mr. W. Trotter, in the 'Pathological Transactions' for 1852; it occurred in St. Mary's Hospital. A young woman, æt. 25, had ulceration of the œsophagus, which extended into the pericardium, and led to sudden syncope and death. For three months she had had nausea, dysphagia, occasional vomiting, and pain at the top of the sternum, and at the epigastrium. Solids were swallowed with much difficulty. There was found, after death, simple ulceration without contraction; the ulcer had extended from the bifurcation of the trachea nearly to the diaphragm, and had perforated the pericardium. No other organ was diseased.

These last two cases were instances of simple ulceration below the bifurcation of the trachea; the other cases were above this part. They appear very similar in character, the modification in the symptoms arising from the difference of the adjoining structures which were implicated.

There are many instances of persons complaining of pain at the upper part of the sternum on swallowing, in whom no trace of pressure or aneurism can be found; and I have seen this symptom disappear under the use of tonics, sometimes with iodide of potassium. The idea of cancerous growth has been precluded; and it has been therefore a question whether some abrasion of the mucous membrane or slight ulceration, such as we sometimes find in the pharynx, had not led to this complaint.

It is exceedingly difficult, during life, to decide as to the character of these fatal cases of ulceration just mentioned; the emaciation, dysphagia, and distress, being the same as in cancerous disease. In all the cases which have come within my notice, the age of the patient has been very much less than in most of those of cancer. This alone, however, is not sufficient to enable us to decide with certainty as to the character of the disease.

The treatment is exceedingly unsatisfactory; the spasmodic contraction of the ulcerated part prevents the passage of œsophageal tubes; no food can be swallowed, and the adminis-



tration of nutrient enemata prolongs life only for a few days or weeks. It is painful to find, after death, that simple ulceration of the œsophagus, or a fistulous communication with the trachea, is the only existing disease; and that if food could have been introduced beyond this point, that life might have been prolonged. The operation of œsophagotomy is a very difficult one, and in many of these cases, if performed, would be quite ineffective, because the disease is often situated at the root of the lung, or behind the first bone of the sternum; in either case, the operation could not be performed below the seat of stricture. It having been found that the peritoneum may be divided without fatal result, and without the terrible effect seen to follow from ruptured viscera, the propriety of forming a gastric fistula in some of these cases is worthy of very serious consideration. It appears certainly warrantable, as it would afford a chance of life to those who now have only a prospect of certain death. In the human subject, several cases of gastric fistulæ accidentally produced have been recorded, and the experimenters on animals purposely make such openings, under the influence of chloroform, without the production of severe peritonitis.

*Cancerous disease of the œsophagus and pharynx.*—Several instances of this disease have been presented at Guy's during the last three years. The symptoms were very similar to those mentioned with ulceration of the œsophagus; the patients are generally beyond the middle period of life; difficulty in swallowing is the first and most prominent symptom, gradually increasing in severity; but in some instances does not become extreme till the extension of the cancerous ulceration to the lungs, or other structures leads to symptoms which almost mask the original disease (as at Case 1). There is pain at the sternum, in the back, sometimes in the upper part of the throat; dyspnœa comes on, where the trachea or bronchi become involved; the dysphagia and emaciation increase, and after six or seven months the disease proves fatal. Sometimes death occurs by inanition, the dysphagia having become complete; more frequently by the extension of the disease to the bronchi, and setting up sloughing pneumonia, or by the pneumogastric nerves becoming destroyed; or, finally, by the ulceration extending into the trachea, and thus leading to fatal

laryngitis. Loss of voice becomes a well-marked symptom only in those cases where the disease is situated at the base of the pharynx, and extending into the larynx at that part. The part of the œsophagus which is most fixed by its connection with other organs, is at the root of the lungs, and it is there that cancerous disease is most frequently found. This, possibly, is on account of its canal in that situation being less yielding, and irritation being the more readily set up.

In all the cases which I have examined, the disease has been of that form which is described as epithelial cancer. The growth presenting modifications of epithelial scales, in the various instances observed, some have been found with very large nuclei; in others, large nuclei were seen thickly set together; in some, brood cells were observed. In Case v, presently to be mentioned, some papillæ were observed on the surface of the growth, covered by healthy squamous epithelium, and containing a capillary filled with blood. See Plate VII, fig. 2 *a*. An adjoining one presented a similar general appearance, but the capillary was filled with granular cells, somewhat resembling white corpuscles of blood. See Plate VII, fig. 2 *b*. A third papilla closely resembled some of the brood cells; its central portion contained nuclei and cells, and it was surrounded by flattened scales or cells resembling epithelium. See Plate VII, fig. 2 *c*. The character of the nucleated cells and epithelium, observed in the same structure, is seen at *d*. It appears probable, that in some cases degeneration of papillæ may lead to the formation of these clusters of cells, and not the endogenous mode of growth which is the method usually received. The disease generally extends by mere contiguity of structure, involving (where any other part is affected, which is not frequently the case,) the adjoining bronchial glands, the roots of the lungs, encroaching upon the bronchi, and setting up pneumonia. I have several times found the pneumogastric nerves destroyed on one or both sides; and it appears that this, in some cases, tends to set up congestion of the lungs, followed by pneumonia, since we find such pneumonia resulting without actual extension into the lung passages.

Case iv presents a marked instance of the disease being found in other structures besides the primary organ affected, cancerous elements being discovered in the liver, pancreas,

stomach, suprarenal capsules, &c. In the liver, lungs, and pancreas, cells of an epithelial character, and precisely similar to those found in the ulcerated œsophagus, were observed. See Plate VII; fig. 1. In this case, also, there was chronic pneumonia existing, not alone, but associated with deposit of small cancerous tubercles. In some cases, a doubt might be felt as to the cancerous character of these bodies, but an instance of this kind removes, I think, such doubt altogether. The vomiting and pain in this case were exceedingly severe, more so than usual, but were explained by the condition of the pneumogastric nerve, which was exposed at the base of the ulcer; several of its branches truncated, and others passing across the surface perfectly exposed. Cancerous infiltration extended around the right semilunar ganglion, and encroached upon its component tissues. All the gland-structures in the abdomen were more or less atrophied; but whether this was due to the diseased condition of the ganglion, or merely dependent on the exhausting fatal disease, and advanced life, is doubtful.

#### CASES OF CANCER OF ŒSOPHAGUS AND PHARYNX, SHOWING THEIR COMPLICATIONS AND TERMINATIONS.

- CASE I.—James R—, æt. 45. Sloughing pneumonia. Pneumogastric involved.
- „ II.—John R—, æt. 50. Communication with trachea. Pneumonia. Diseased kidneys.
- „ III.—George E—, æt. 73. Gangrene of lung. Cancer of cervical gland and thyroid body.
- „ IV.—Jane B—, æt. 63. Cancer of stomach, liver, pancreas. Chronic pneumonia with cancer. Destruction of pneumogastric. Granular kidneys. Diseased semi-lunar ganglion.
- „ V.—Charlotte W—, æt. 32. Disease at termination of pharynx. Laryngitis.
- „ VI.—Catherine S—, æt. 38. Communication with trachea. Cancer of lung and kidney.
- „ VII.—Mrs. B—, æt. 54. Death from inanition.
- „ VIII.—George D—, æt. 45. Gangrene of the lung.



CASE IX.—John H—, æt. 66. Pleurisy and diseased kidneys.  
 „ x.—Martha M—, æt. 31. Cancer of palate, with  
 strumous pneumonia.

CASE I.—*Cancer of the œsophagus. Sloughing pneumonia, the pneumogastric involved.*

James R—, æt. 45, admitted into Guy's, November 21st, 1854, under Dr. Gull's care. He died November 30th. He was a married man, a labourer, but had not been temperate in his habits. Nine weeks before admission he was unable to swallow his food with comfort, and he suffered from severe pain at the lower part of the sternum. After that time he lost flesh much, and cough, with pain in his side, came on. He vomited occasionally, and had burning pain at the sternum; and there was a sense of nausea when he began to eat. On admission, he had a cachectic, pale, and wretched appearance; he was troubled with cough, and the expectorated matters were exceedingly offensive. At the left apex the respiration was coarse; but at the base of the right lung there were signs of consolidation; the voice, both at the base and apices, was increased in resonance. Cinchona and morphia were administered, but the patient sank in a few days. The severe pulmonary symptoms in this case completely masked the original disease of the œsophagus; for a short time it was believed that it was a case of pneumonia with old disease of the lung, and that the burning pain at the sternum, and vomiting, were consequent on intemperate habits. At the commencement of the œsophagus was an extensive ulceration, four or five inches in length, irregularly tubercular on its surface, and several tubercles were situated in the mucous membrane, both above and below the ulceration. The disease extended as low as the root of the lung, but the lungs themselves and the pleura were free from cancerous disease. The tissue external to the œsophagus was extensively infiltrated, especially on the right side, and some of the bronchial glands affected; the right pneumogastric nerve extended through it. The lower part of the pneumogastric appeared wasted, but it could not be traced satisfactorily throughout, having been divided in the inspection. The right lung, at its lower lobe, was of a greenish colour, and of a faint gangrenous odour, infiltrated with dirty serum, and imperfectly consolidated. The bronchi were intensely congested. The remaining parts of the lungs and larynx were healthy. The heart, stomach, liver, and intestines, &c., were healthy; and no cancerous disease could be detected in any other part. As to the character of the growth, it had the general and microscopical appearance of epithelial cancer. There was no direct communication between any of the large bronchi and the ulceration of the œsophagus; and it appeared probable that the right pneumogastric, becoming involved in the disease, had predisposed to the pneumonic inflammation on the same side. The disease proved fatal at an earlier period than usually observed, only about ten weeks from the recorded commencement of difficulty in swallowing.

CASE II.—*Epithelial cancer of the œsophagus communicating with the trachea. Pneumonia. Granular kidneys.*

John R—, æt. 50, admitted into Guy's, under Mr. Hilton's care, March, 1856, and died the following day. No history, except that he had felt ill for three



months, could be obtained. On admission he was suffering urgent dyspnœa, there was great congestion of the face, and he was apparently dying from apnœa. Tracheotomy was performed by Mr. Callaway, but the patient died in a few hours. The body was spare, but muscular. The epiglottis was pale, and its mucous membrane slightly œdematous; near the arytenoid cartilage was a small circular ulcer. At the commencement of the trachea was the artificial opening, and two inches and a quarter below it, about an inch above the bifurcation, was a vertical opening about half an inch in length, extending into the œsophagus; the membrane around was of a dull gray colour; the mucous membrane of the trachea and bronchi were much congested. About three inches above the commencement of the œsophagus was an irregular ulcer, three inches in length, with raised, irregular edges, and ragged surface; at its base was the opening into the trachea; the remaining part of the canal was healthy. (Preparation 1793<sup>34</sup>.) Neither bronchial nor cervical glands were affected. The lower lobe of the right lung was in a state of red hepatization, becoming gray; and in the left lung was a lobule broken down from acute inflammation. On the right pleura there was effusion of lymph. The kidneys were small, granular, contracted, and containing cysts. The other viscera healthy.

CASE III.—*Cancer of the œsophagus, of cervical glands, and of thyroid body. Gangrene of the lung.*

George E—, æt. 73, admitted into Guy's, November, 1853, and died February, 1854. He was a table-cover maker, and in his early life had been intemperate; he was extremely emaciated. Eight months before admission he received a severe fall, from which he never recovered; and two months later he began to suffer great pain in eating solids, and had occasional attacks of vomiting. These became more and more frequent, and latterly almost incessant. He could not take solid food, and complained of intense pain at the cardiac extremity of the stomach. Mr. Callaway passed an œsophageal bougie, but without meeting with any obstruction in its passage. His vomiting diminished soon after admission. The bowels became constipated, but he continued to suffer severe pain. He became gradually weaker, and, on February 5th, vomited a considerable quantity of dark-coloured fluid; he died on the 13th. Inspection was made twenty hours after death. In the brain there was considerable subarachnoid effusion, and disease of the arteries. At the central part of the œsophagus, opposite the root of the lung, was a large irregular ulcer, two inches in length, and involving the whole of the tube; at the upper part was a raised, circular margin, and a semi-detached ulcer of similar character, about half an inch in diameter. At the root of the right lung was a mass of sloughing tissue, infiltrated with sanious fluid, and the adjoining lung was consolidated. At the base of the left lung was a circumscribed mass of pulmonary apoplexy and lobular pneumonia, and a vomica containing thin purulent fluid. The cervical glands and the thyroid body were infiltrated with carcinomatous product, white, and resembling medullary cancer. There was fatty and fibroid degeneration of the heart. In the peritoneum were old adhesions, and a granular condition of the surface of the liver. The kidneys were also granular and contracted.

CASE IV.—*Epithelial cancer of œsophagus. Pancreas. Liver. Stomach. The pneumogastric involved. Granular kidneys. Chronic pleuro-pneumonia, with cancer. Fibrous tumour in uterus. Cancer of suprarenal capsules and semilunar ganglion.*

Jane B—, æt. 63, admitted August 23d, 1855, under Dr. Addison's care. She was a married woman, who had resided at Snows Fields, and had had three children. She was of light complexion; a thin anæmiated woman. She had suffered for nine months; the first symptom being pain after swallowing; no tumour could then be felt, but cancerous disease was suspected. After admission, at the scrobiculus cordis, a firm mass, about the size of a hen's egg, could be felt; it was well defined, sensitive on pressure, and tolerably distinct pulsation could be felt in it; the food returned at once, or rather was at once regurgitated. The tongue was clean, and the bowels constipated. She complained much of flatulence, and at night regurgitated water into the mouth. At first, vomiting several hours after food was the principal symptom. Soon after admission the food was at once returned; sometimes, however, it was retained for several days. She took creosote three times a day, and opium at night, with considerable relief for a short time. On December 8th, I examined some of the water ejected from the mouth, but could not discover any cancer-cells or sarcina. She varied much, sometimes the stomach being excessively irritable, and rejecting everything, at other times she was able to take food. On December 19th, tumour had not increased in size. She became more and more prostrate and during the last month of her life suffered severely. She died March 26th. *On inspection*, the body was much emaciated. The brain was very much atrophied; the convolutions separate. There was subarachnoid effusion, and an increase of fluid in the ventricles themselves. The septum in the ventricles was atrophied, and almost destroyed. *Chest*.—At the commencement of the œsophagus, the mucous membrane began to present an irregular granular appearance, with one or two whitish tubercles about the size of pins' heads; passing downwards, these became more numerous, till nearly opposite the root of the lung a raised ulcerated margin was observed, and this was in some parts sloughy; beneath this, the walls of the œsophagus were completely destroyed for about three inches, and the side of the right lung was in a sloughy condition; posteriorly, the pericardium bounded this sloughy mass, and there was an opening about the size of a sixpence extending through that membrane, opposite the left auricle, which was slightly affected with granular cancerous growth at that part. Nearer to the stomach, the walls of the œsophagus were again observed intact, but infiltrated with cancerous product, and nearly in a sloughy condition. At the floor of the cancerous ulcer were several branches of the pneumogastric exposed; the right one could be traced down to the ulcer, and several branches were completely truncated; another one passed obliquely across the ulcer to the opposite side, to join the left nerve. On the left side, a branch was also observed to be truncated, and a large one ran for about two inches exposed in the sloughy tissue. The branches to the lungs were entire above the cancerous growth. The ulcer in the œsophagus presented the elements of epithelial cancer. At the left lung were pleural adhesions; and the apex presented several white tubercles, at first supposed to be strumous, but found to consist of cancerous elements; the surrounding lung and the whole apex

were of an iron-gray colour, from chronic pneumonia. The microscope showed large nucleated cells, resembling those of epithelial cancer observed in other parts, smaller nucleated cells, a great number of granules, pigment, and some elongated fibre cells; other tubercles of a similar kind were observed in the lung, a few near the root, others at the periphery; the right lung was more free from disease. The larynx, trachea, and bronchi were healthy. The heart was small, destitute of fat, and atrophied. *Abdomen*: In the stomach, near the œsophageal opening, was a raised tubercular growth about half an inch in diameter; it was ulcerated at its apex; its section showed that it principally involved the mucous membrane, but was extending into the muscular coat beneath. Some large nucleated cells were observed in the raised edges of the growth, and degenerated gastric follicles, some much enlarged, and containing highly refracting particles, others nuclei. The rest of the mucous membrane and the pylorus were healthy. The head of the pancreas formed the hard mass which had been felt at the scrobiculus cordis; it consisted of hard, granular, whitish tissue, soft and breaking down in the centre, with some dense, firm, semi-transparent bands passing through it. On examination, it presented large epithelial cancer cells, elongated cells forming fibres, and some undergoing degeneration (see Plate VII, fig. 1 *b*). The adjoining glands were infiltrated and adherent, the lesser curvature of the stomach was also adherent; the rest of the pancreas was normal. On the adjoining surface of the liver was an irregular tubercle, evidently produced by contact, and in the substance were several other small tubercles, but consisting of the same epithelial elements (see Plate VII, fig. 1 *a*). The liver was small; its cell structure healthy. The gall-bladder was moderately distended, so also some of the bile ducts. On the right side, the cancerous infiltration extended to the right semi-lunar ganglion, which appeared to be infiltrated with cancerous product; cancerous cells being observed among the ganglionic cells. There were cancerous tubercles in both suprarenal capsules, but only involving a small portion of the organ. The duodenum, ileum, and colon were healthy, but the intestines were atrophied, thin, and wasted. The kidneys were granular, very small, and only four ounces in weight. The spleen was small, and there was a dense white patch on its surface. The cavity of the uterus was occupied by two soft polypi, and a large dense tumour, about three inches in diameter, involved its walls; it was dense and fibrous at its periphery, but did not present any trace of cancerous elements; its centre was tough, gray, and semicalcareous; the ovaries atrophied. (See Preparation 1793<sup>33</sup>).

*CASE V.—Epithelial cancerous tumour in pharynx, closing entrance into œsophagus, and in the neck. Effusion of false membrane in the larynx and trachea. Acute bronchitis.*

Charlotte W—, æt. 32, admitted under Mr. Cock's care, February, 1856, and died March 6th. She was an anæmiated, short, married woman, somewhat emaciated. She had been out of health for a year, but for three months experienced very great difficulty in swallowing, and for several days it had become almost impossible to swallow anything except a small quantity of fluid; and the attempt now led to regurgitation of it through the nares. The attempt to swallow did not produce urgent dyspnœa. Respiration on admission was easy and normal, but there was slight hoarseness. On examining the chest, the respiration was found to be less free at the right apex. At the left side of the neck below the angle of the



jaw was a prominent round tumour about one inch in diameter; it could be partially separated from the structures beneath. Mr. Cock attempted to pass a small bougie, but it was found to be quite impossible. The tumour could not be seen in the throat or felt. A short time before death very urgent dyspnœa came on, and she died from apnœa. At the lower part of the pharynx, attached to the cricoid and arytenoid cartilages, or rather the mucous membrane opposed to them, were four round tumours closely placed together, or rather one lobulated growth, extending as high as the upper margin of the epiglottis, and quite excluding the opening into the œsophagus. After removal, a probe could only be inserted by slowly passing it round the growth. The surface of the growth was gray, not ulcerated; its section pale, and nearer the surface presented regular red lines; vessels full of blood. The soft palate also was considerably thickened. The inner surface of the epiglottis, of the larynx, and of the trachea, was covered by a layer of false membrane easily separable; the bronchi, especially the larger ones, were also full of tenacious mucus. The tumour in the neck was soft, and of a pale yellow colour. The lungs did not collapse, but appeared quite healthy. Heart, liver, intestines, spleen, and kidneys, were healthy; so also the uterus and ovaries. Bronchial and abdominal glands normal. On examining the growth from the pharynx, its base was found to consist of large cancer-cells, containing a large granular nucleus, and closely arranged together. (See Plate VII, fig. 2 *d.*) The growth in the neck had a similar structure. The surface was not ulcerated, but presented epithelium, normal in some parts. The appearance of the papillæ has been previously referred to; some were in a normal condition; in others, the central capillary was obstructed, and some were still more degenerated, closely resembling brood cells. (Fig. *a, b, c.*) (Prep. 1785<sup>76</sup>.)

CASE VI.—*Carcinoma of œsophagus, communicating with the trachea. Cancer of lung and kidney.*

Catherine S—, æt. 38, admitted under Dr. Barlow's care, April 9th, 1856, and died April 17th. She had been a servant in a family for twenty years, and began to suffer from her present illness about six months before her death. On admission, she was in a state of extreme emaciation, the dysphagia was extreme. Swallowing of food was at once followed by its regurgitation through the nose and mouth. The circulation was exceedingly feeble, and Dr. Barlow feared lest gangrene in the extremities might come on. She appeared to die from exhaustion. The body was much emaciated. In the neck on the left side was an enlarged cervical gland, about one inch in diameter, firmly adherent to the œsophagus and trachea; a smaller gland was situated on the right side; the former could be felt before the division of the skin. The lungs did not collapse freely. On dividing the trachea an opening was found immediately above the division into the bronchi, somewhat oval in form, slightly pointed above and below, and about one inch and a half long, communicating with the œsophagus; the edges of this opening were thickened and slightly irregular. The corresponding part of the œsophagus presented a nodular surface about three inches in length, and involving the whole circumference of the tube. The edges were raised and irregular, and the surface ulcerating. There was slight vascular turgescence. Several cervical glands were adherent to the œsophagus, were of a firm white colour, and infiltrated with cancerous deposit, and in the centre of a yellow



colour. Other glands at the root of the lung were not at all infiltrated. The bronchi were intensely congested, and contained much dirty grumous fluid. The lower lobes of the lungs were much congested, and the right contained beneath the pleura a small mass, about half an inch long and a quarter of an inch broad, composed of yellowish-white cancerous substance. The left renal vein was filled with clot, which was adherent, and the walls were considerably thickened. In this kidney were several cysts, and a minute tubercle composed of elements resembling the other cancerous structures. On examination of the œsophageal ulcer, a small quantity of juice from the section presented numerous nuclei; and in the section, some epithelial plates, cells with large nuclei, and caudate cells. It also presented some elongated nuclei and fibres, some of which had a curved arrangement inclosing nuclei and brood cells. The raised edges of the ulcer were composed of masses of these nuclei and cells, with some intervening elongated nuclei and fibres; and, on the addition of acetic acid, some elastic coiled fibres were observed. The growth in the lung presented similar aggregation of nuclei. The cervical glands were of a much firmer texture, and much fibrous tissue was observed, forming irregular interspaces, in which nuclei were found. The central portions were yellow, and contained much fat (degenerating cancer). The great number of large nuclei resembled those found in medullary cancer, and this case appeared to be almost intermediate between medullary and epithelial disease.

A brief mention of some other cases recorded in the post-mortem inspections at Guy's will serve to illustrate some of the points previously mentioned.

#### CASE VII.—*Cancer of the œsophagus. Death from exhaustion.*

Mrs. B—, æt. 54. A stout married woman, who had ten children, and ceased to menstruate for four years, experienced seven months before her death pain at the middle of the sternum, at the scapula and loins; the pain was increased on taking food; she had slight palpitation of the heart, vertigo, and flatulence; her sleep was disturbed by pain; passing a probang down the œsophagus much increased the pain. This became very severe, and was aggravated by a chronic winter cough; deglutition became more difficult, nutrient enemata were used, but she gradually sank, seven months after the commencement of the dysphagia. The central portion of the œsophagus was converted into a softened discoloured brain-like substance, from two and a half to three inches in extent. No gland or other structure was affected, and the remaining part of the tube was healthy.

#### CASE VIII.—*Cancer of œsophagus. Gangrene of the lung.*

George D—, æt. 45, a very intemperate man of irregular habits, was admitted into Guy's, suffering from symptoms of stricture of the œsophagus, with supposed phthisis. The breath was exceedingly offensive. At the apex of the left lung was a large gangrenous cavity. There was extensive ulceration of the œsophagus, which had ulcerated through to the vertebra, and communicated with the left bronchus. The sternal and mesenteric glands were enlarged.

CASE IX.—*Cancer of œsophagus, with pleurisy and contracted kidneys.*

John H—, æt. 66, seven months before his death first experienced difficulty in swallowing solids. The dysphagia increased, and swallowing at last became impossible. Till two days before his death he had no pain in his throat, but severe pain in the left side; this arose from pleurisy, the right pleura being found to contain more than a pint of purulent serum after death. There was constriction of the œsophagus, with some ulceration and considerable thickening around it. The kidneys were granular.

CASE X.—*Carcinoma of throat. Strumous pneumonia.*

Martha M—, æt. 31, admitted December 5th, 1855, under Dr. Addison's care, and died on the 20th, at two p.m. She was a short woman, married, and had been confined fourteen months previously, but since that time had not been well, having suffered from a slight cough. For three weeks she had had difficulty in swallowing, and this had increased to such an extent that she was on admission unable to swallow food, except with extreme difficulty. She could, with much distressing pain, swallow solids, but fluids at once regurgitated through the nose. She suffered from hunger, but still more from thirst. She was extremely emaciated. The glands at the angle of the jaw on the right side were much enlarged, giving her emaciated countenance a miserable appearance. Her voice was nasal, and she was extremely exhausted. She was too ill to allow the chest to be examined, and died on the 20th. Her relatives, brother, &c., died of phthisis. On removing the larynx and tongue the soft palate was found to be about twice its natural thickness, irregularly tubercular, and brawny; the posterior pillars of the fauces were affected in a similar manner (Preparation 17857<sup>o</sup>). On the right side there was a communication from the pharynx into an irregular cavity, situated opposite or rather behind the angle of the jaw, about two inches and a half in length, and half an inch in breadth, and containing almost black, sloughy substance. The glands were infiltrated with firm, cancerous product. The tissue of which the soft palate was composed consisted of an immense number of nuclei. In the lungs, there were firm adhesions at the apex of the right lung, the pleura being semicartilaginous. In the remaining part of the lung numerous minute tubercles were observed beneath the pleura, and at the lower lobe there were also moderately firm adhesions. The left pleura was free. At the apex of the right lung was an irregular vomica, capable of holding about two drachms of fluid, with a smooth lining, and surrounded by iron-gray lung, and several opaque tubercles. At the lower lobe a considerable portion of the lung was red and consolidated, and several lobules infiltrated with pale yellow, low organized deposit, breaking down in several parts, and precisely resembling the lung observed in cases of acute pulmonary phthisis. There was considerable congestion of the bronchi, and tenacious mucus in them. The left lung was congested, but otherwise healthy. The bronchial glands were black from pigment, and those quite at the base of the neck were firm, white, and dense, consisting of nuclei resembling those in the palate. In the lung, the tubercles presented no nuclei resembling those in the diseased palate, nor

consisted of cancerous growths, but were composed of imperfectly developed nuclei, dark, pigmental granules, and some nucleated cells. The tissue of the thickened pleura consisted of fibrous tissue. *Heart* exceedingly small, destitute of fat; its cavities contained moderately firm clot, and the valves were healthy. *Liver* healthy, so also the spleen. Stomach and intestines contracted; healthy, except the rectum, and sigmoid flexure, the mucous membranes of which were congested in longitudinal stripes, and numerous minute superficial ulcers were scattered along these patches.

This case is one of great interest in the connection of cancerous disease of the pharynx with strumous pneumonia—diseases rarely conjoined, occurring at different periods of life, and considered antagonistic the one to the other. In advanced life, where death has occurred from cancer, we sometimes find the remains of strumous disease at any earlier period of life, calcareous degenerated glands, or such a mass at the apex of the lung, surrounded by dense lung tissue; such cases are, however, rare and exceptional.

*Obstruction of the œsophagus from pressure of aneurismal or other tumours.*—The œsophagus is in close contact with the aorta, and we frequently find that dysphagia is one of the symptoms of aneurismal dilatation of that vessel. It is no uncommon thing to find death suddenly taking place from rupture of the aneurism into that canal, although, in many instances, death may arise from different causes, even if there has been considerable pressure, and sloughing of the œsophagus produced. The pain, dyspnœa, and dysphagia, in some of these cases, is much relieved when the patient bends the body forward, so as to remove the pressure from the structures beneath. In instances such as these, we have other indications of the nature of the disease, neither is the emaciation so great as we find in previously mentioned cases, although the paroxysms of dyspnœa and pain are exceedingly severe.

#### *Aneurism of the aorta and sloughing œsophagus.*

James F—, æt. 34, was admitted, under Dr. Hughes's care, November, 1855, and died in January, 1856; he was a married, temperate man, a labourer at Dartford. Six months before his admission, after having been engaged a short time previously in carrying very heavy weights, he experienced pain in the left breast; this pain became much more severe, and also extended between his shoulders, but there was no tenderness in the back. On December 4th the pain at the left nipple became more fixed, and there was a slight systolic *bruit*. On January 1st, Dr. Hughes



noticed that the radial pulse was weaker on the right side, and he was found to have difficulty in swallowing solids. This dysphagia increased in severity, and his dyspnoea became more distressing. On January 20th he was unable to swallow food; his face was livid, dyspnoea urgent, and his pain severe. He died on the 25th. On examining the chest, the lungs were emphysematous, pale, but moderately collapsed. There was acute inflammation of the pericardium, and considerable injection of the pleura on both sides. On turning aside the lungs, an aneurismal tumour, about the size of a large orange, was found at the termination of the arch of the aorta; its walls were thin; the posterior part of the vessel was entirely destroyed, and communicated with a cavity in front of the vertebræ, one of which was absorbed. There was scarcely any fibrin in the sac. The aneurismal tumour had pressed upon the œsophagus, and quite obliterated its canal; the whole of its walls were of a greenish colour, very offensive, and in a sloughing condition. Still no perforation had taken place. Both bronchi were compressed. Two other aneurismal tumours were found connected with the ascending and transverse portions of the arch of the aorta. Other viscera were healthy.

*Aneurism of ascending aorta rupturing into pericardium.  
Communication of œsophagus with left bronchus.*

Frederick K—, æt. 23, admitted under Dr. Gull's care, January 23d, and died April 26th, 1856. He was a hawker, and had been living in the Old Kent Road; he had enjoyed good health till five months ago, when he struck his chest against a box hanging from a crane; a fortnight afterwards, he experienced pain at the part; this gradually increased till three weeks before admission, when he was obliged to give up work. On admission, he complained of pain in his chest, a distinct pulsation could be felt between the second and third ribs on the right side, and a jar with the second sound of the heart. There was pain at the seat of pulsation, and along the border of the pectoralis major, and down the inner side of the arm. The pain continued severe, and a systolic bruit became audible at the seat of the tumour. He could obtain no rest at night. On April 19th, he had difficulty in swallowing, and this increased much in severity. On the 28th, after talking with his friends, he died very suddenly. On removing the sternum, an aneurism of the ascending aorta was opened, it had extended to the sternum on the right side. On further opening the pericardium, it was discovered to be full of blood, and a small irregular opening was found at its upper part into the aorta. The heart was of normal size; the left ventricle not hypertrophied; the valves healthy. The ascending aorta formed an aneurismal sac, about two inches and a half in diameter, principally on the right side. The lung was adherent, and it was nearly perforated. The aneurism extended as far as the left carotid; below the left subclavian was another small dilatation. At the centre of the œsophagus, where it is near the left bronchus, was a slough, and an opening into the bronchus, with considerable inflammatory tissue. No communication, however, with the vessel. The remaining viscera were healthy. Although it appeared that the greatest pressure from the aneurism was on the right rather than the left side, we can find no other explanation for this sloughing condition of the œsophagus, and its communication with the bronchus, beside the pressure which all these parts suffered from the distended aneurismal sac.



Cases of this kind should render us exceedingly cautious in the use of œsophageal bougies, lest they lead to the sudden rupture of an aneurism, and the death of the patient.

In studying the diseases of the œsophagus, *gastric solution* of its lower extremity must be borne in mind. This subject has been very clearly brought forward in the communications to the 'Guy's Reports,' by Mr. Wilkinson King, in the years 1842 and 1843. It is exceedingly frequent to find the mucous membrane of the œsophagus abruptly terminating at the cardiac extremity of the stomach, from the solvent action of the gastric juice having extended to that line; but on opening the canal of the œsophagus itself, for several inches, near its lower extremity, the upper margins of the rugæ are often found deprived of mucous membrane, and long shreds are observed on stretching out the tube, the parts which have escaped digestion. This solution extends into the mediastinum, as found in cases mentioned in the communication just referred to, or into the pleura itself, the contents of the stomach escaping into the left pleural cavity, that which is in closer relation with the œsophagus.

Only two cases of this perforation of the œsophagus have occurred at Guy's during the last three years—one in a case of fever, another hydrocephalus—so that it is a circumstance of unfrequent occurrence. The causes which lead to solution of the stomach are more clearly understood than formerly. Dr. Budd has very lucidly, in his work on 'Diseases of the Stomach,' brought forward all that is known on the subject. The position of the body, the development of gases in the intestines pressing upon the contents of the stomach, the non-contracted state of the œsophagus itself, are causes which produce the passage of the gastric juice into the œsophagus. Sometimes, indeed, this pressure forces the contents into the pharynx, and we find them gravitating into the trachea and bronchi.

Amongst other affections connected with dysphagia is—

*Ecchymosis*.—Hemorrhage from the œsophagus generally arises from the rupture of aneurismal tumours, or from cancerous disease; but in cases of fatal purpura, we sometimes find the whole mucous membrane covered by points of effused blood, and blood is also effused into the surrounding cellular

tissue. This part, however, is affected only in common with the whole mucous surface of the alimentary canal, as well as with other membranes and gland-structures.

The formation of polypi, and the extension of disease from the vertebræ into the pharynx and œsophagus, will be found described by surgical authors, to whom I must refer for their description.

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### PLATE VI.

#### ILLUSTRATING DR. HABERSHON'S OBSERVATIONS ON THE SYMPATHETIC NERVE.

Dissection of the semilunar ganglia and solar plexus, and of the phrenic and pneumogastric nerves, seen from the vertebral aspect; the aorta, &c., being removed.

- a.* Right pneumogastric.
- b.* Left pneumogastric.
- c.* Branch from left pneumogastric, sending a filament to the suprarenal capsule, one to join the semilunar ganglion, and another to the phrenic.
- d.* Right semilunar ganglion.
- e.* Left semilunar ganglion.
- f.* Diaphragmatic ganglion.
- g.* Branch from it to join the phrenic.
- h.* Branches to anterior surface of the lobulus spigelii.
- i i.* Phrenic nerves.
- k k.* Diaphragmatic branches of phrenic.
- l.* Branch from left phrenic to join pneumogastric and semilunar ganglion.
- m m.* Greater splanchnic nerves.
- n n.* Lesser splanchnic nerves.
- o.* Celiac axis.
- p.* Superior mesenteric artery.
- r r.* Renal arteries.
- s s.* Renal plexus of nerves.
- t t.* Branches in front of the aorta.
- v v.* Branches to suprarenal capsules, on right side, forming two loops in their substance.
- x x.* Lobulus spigelii and liver.
- y y.* Stomach.
- z.* Pancreas.









## PLATE VII.

Figures 1 and 2 illustrate Dr. Habershon's cases of cancer of the œsophagus.

Fig. 1 exhibits the microscopic appearance of the epithelial cancer, in Case iv, p. 224, magnified about 400 diameters.

- a.* Epithelial-cancer cells from the liver.
- b.* Epithelial-cancer cells from the pancreas.

Fig. 2 exhibits the microscopic appearance of the epithelial cancer, in Case v, p. 225, magnified about 400 diameters.

- a.* A papilla, containing a capillary vessel filled with blood, and surrounded by nearly healthy epithelium.
- b.* A similar papilla, filled with white corpuscles, and surrounded by epithelium in a state of transition to cancer.
- c.* A papilla still more degenerated, and approaching in its structure more nearly to the character of cancer.
- d.* Epithelium from the surface of the ulcer of the cancerous growth in the pharynx.

Fig. 3 shows Dr. Habershon's dissection of the diaphragmatic ganglion, and the branches to it from the pneumogastric and phrenic nerves, and semilunar ganglion.

- a.* Pneumogastric nerve.
- b.* Diaphragmatic ganglion situated on the vena cava.
- c.* Phrenic nerve.
- d.* Diaphragmatic branches of nerves.
- e.* Splanchnic nerve.
- f.* Stomach.
- g.* Coronary artery.
- h.* Celiac axis.
- i.* Vena cava.
- k k.* Diaphragm.

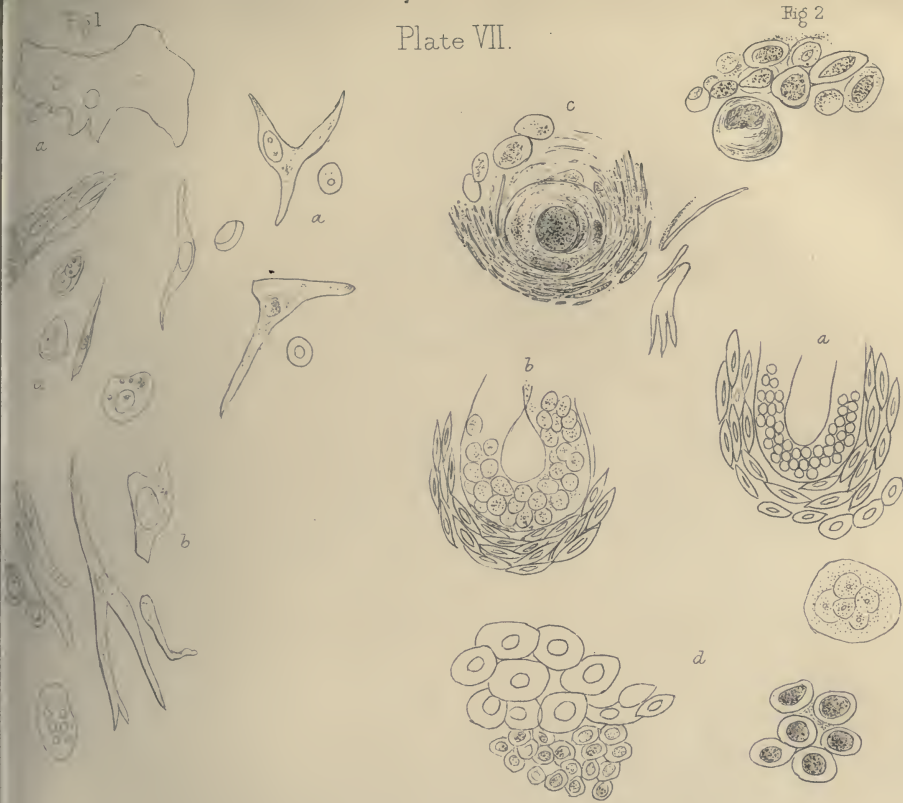


Fig 3.







ON THE USE  
OF  
ATROPINE IN IRITIS.

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BY JOHN F. FRANCE.

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I PROPOSE in the succeeding remarks to consider the beneficial effect of belladonna and its alkaloid, atropine, in inflammation of the iris. It will seem a work of supererogation to most surgeons having practical knowledge of this disease to affirm and adduce proof of that which they may well regard as an ascertained fact. Nor should I have conceived the idea of doing so, but that recently, by an able and generally judicious writer, the remedial power of belladonna in iritis has been impugned, and its use represented as injurious. Under these circumstances, looking to the importance of the question in practice, and the impossibility of determining it except by that appeal to experience which can only be pursued satisfactorily where a large ophthalmic constituency is available, I have considered it quite consistent with the design and character of this work to enter upon the subject, with the view of controverting a grave therapeutical error.

The action of belladonna in health is, of course, beyond dispute; every tyro in medicine is acquainted with the fact that it causes the pupil to expand. It is equally demonstrable that by this change the iris is removed from the capsule of the lens; for the convex form of the latter structure bringing its central portion forward into immediate proximity with the plane of the iris (and consequently into close juxtaposition with the margin of the pupil when contracted), creates towards the periphery (and therefore in the dilated state of pupil) a comparatively wide interval between the same parts.

These incontestable effects upon the healthy eye are the grounds on which the use of belladonna in iritis is advocated, because in this disease the pupillary aperture is threatened

with permanent diminution or closure from the encroachment of inflammatory exudation. Now it requires few words to prove that a large pupillary area is, *cæteris paribus*, less likely to be wholly or in great proportion covered with lymph than one of small dimensions; and, again, that a pupillary margin removed to a considerable distance from the crystalline capsule is less likely to adhere thereto than one already nearly or quite in contact with it. So conclusive do these simple facts appear in favour of procuring dilatation of pupil when the iris has become the seat of inflammation, that the practice of applying belladonna for the purpose has been long universally adopted. Such advantageous uniformity of practice, however, may well be disturbed (among those at least who have only occasional opportunities of witnessing eye diseases), if the dictum, *ex cathedra*, that the approved remedy is useless in the earlier period of iritis, injurious in the later, be suffered to pass unchallenged. That it deserves not this character, but is, on the contrary, both an efficient agent and a purely beneficial one, long and careful observation entitle me to affirm. I will proceed, therefore, to examine the basis on which this novel objection rests.

First, it is stated that an inflamed iris loses its power of motion, and that atropine, therefore, must be useless during the active stage of inflammation.<sup>1</sup> Here a general conclusion is drawn from a proposition only partially correct. An inflamed iris does indeed lose its power of motion when the disease has united it by thick glutinous lymph to the adjacent capsule: it loses its power of motion, too, if similar exudation have infiltrated its texture,—coextensively with that infiltration. This is freely granted. But happily in the majority of instances we are consulted before these changes have been accomplished, before fibrin has been effused in any great quantity, either on the surface or margin of the iris, or into its parenchyma; before it has ceased to respond, in some degree at least, to its natural stimulus, and to obey the artificial influence of belladonna. It would not be difficult to adduce numerous cases in proof of this, for it is the familiar result of experience. I will not, however, fatigue the reader by their recital; but content myself with two recent examples.

<sup>1</sup> Dixon on 'Diseases of the Eye,' p. 138, 1855.

CASE I.—Michael D—, æt. 28, on return from the Crimea, contracted syphilis at Chatham, and on appearing at Guy's, on August 5th, was the subject of syphilitic lichen, and iritis on both sides. The latter disease had attacked the right eye ten days, the left six days previously, and both organs had in the interval become progressively worse. The right eye presented the usual sclerotic zone, masked by conjunctival vascularity: the cornea was bright and clear: the iris dull and discoloured, of a greenish hue; the pupil circular, contracted, and restricted in activity, but still responding in an appreciable degree to light; its area not suffused, but vision very dim, as if obstructed by a thick fog. The left eye exhibited precisely the same appearances in a milder degree, the pupil, however, being less contracted, but misshapen from adhesion. An hour or two after atropine<sup>1</sup> had been used, the right pupil had expanded to nearly double its former diameter, and become irregular, from dark brown adhesions which now came into view. The left pupil opened widely in the horizontal direction, especially at the temporal side, while above and below adhesion limited its expansion. The man was cupped on the right temple; but, by a mistake, the internal remedies ordered were not exhibited until after the next day's visit. In the mean time, however, both pupils had yielded still more to the action of the belladonna (which was kept applied in extract), and had become well dilated, the right pupil to more than double its former diameter, and the left more completely still.

CASE II.—William S—, æt. 22, a smith, was admitted into Guy's on August 8th. He acknowledged having been the subject of primary syphilis eight months previously, which was followed by secondary eruption, but not by disease of the eye until six days before application. At that time, the left eye inflamed, and grew progressively worse, the only medicines employed being two pills and a purgative draught. On presenting himself at Guy's, he manifested the usual symptoms of acute iritis; there was considerable zonular sclerotic injection, with dulness and discoloration of the iris, a con-

<sup>1</sup> In the proportion of three grains of the sulphate to the ounce of water.



tracted but circular, and apparently clear pupil; the vision was much impaired; the right eye was sound. An hour after atropine solution had been dropped on the conjunctiva, the left pupil had expanded to more than twice its former area, and become vertically elongated, owing to dark lateral adhesions limiting its dilatation in the horizontal direction. The adhesions, being quite recent, were weak, and had apparently yielded by the next day, when the pupil was found still more dilated, and no longer of the marked oblong form. The usual treatment had in the mean time commenced.

Both patients left the hospital convalescent in three weeks.

The complete responsiveness of the iris, which exists in the earliest stage of inflammation, becomes modified as the disease advances. The pupil will then expand more tardily, and to a less degree; until, when copious effusion has taken place, it may, as above mentioned, become powerless to act from mere mechanical encumbrance. Up to this stage, however, every gradation of mobility may be met with; and often, even when bulky lymphatic exudation has occurred (if it be but circumscribed in nodules or tubercles, as in well-marked syphilitic cases), atropine will still produce notable dilatation in the intervals.

If this be so (and let any doubting reader simply try the experiment for himself in the first half-dozen cases he encounters), the objection I am combating falls to the ground; the effect of atropine is *not* negative, and we may avail ourselves of its aid, in every case. In those which respond to its action we shall have gained an important advantage in the way explained at the outset, by preventing or limiting adhesion and suffusion of the pupil; those which resist its influence simply remain as before not benefited indeed (for the time at least), by the application, but in no respect injured by it.

For, the second objection to the use of belladonna has as slender a foundation as the first. It is this, that in the later period of iritis, when it is admitted that the iris is recovering its motory functions, atropine may do harm, by forcibly dilating the pupil, rupturing the newly formed adhesions, and leaving patches of lymph coloured by uveal pigment



upon the capsule. All agree that the active principle of belladonna possesses this quality of producing forcible dilatation. The energy of its action is often made apparent by the deep festoons or bays into which the free portions of a partially adherent iris are drawn. There is evidently a strong retractile power exerted by the radiant fibres; so that the shape of the pupillary area is often made to resemble an architectural trefoil, quatrefoil, or cinquefoil; of which the crescentic compartments are represented by dilated portions of the margin, the cusps by the points of adhesion. By perseverance with belladonna, and simultaneous employment of appropriate internal remedies, adhesions of the character just described will not unfrequently give way, the iris recovering its freedom to a proportionate extent, and sometimes the point of capsule corresponding to each late adhesion presenting, on close scrutiny, for a longer or shorter period, a minute brown patch. It is noticeable that these phenomena are especially met with in syphilitic cases, in which adhesions are commonly filamentous, and discoloured by pigment. The adhesions occurring in rheumatic and other kinds of iritis are, for the most part, whitish, and destitute of pigment; and, being in the form of broader bands, their separation is comparatively seldom accomplished. Hence, in practice, the only cases in which release of an adherent iris, and relinquishment of pigment marks are probable, are those wherein the adhesions consist of narrow threads, and the sites of attachment are mere points.

Now, in what way can the isolation of minute brown patches upon the capsule corresponding with such points be otherwise than a great gain, when compared with their previous state in connection and continuity with adhesions? While the pupil remains attached, not only is the brown spot equally resident on the capsule, but it constitutes a fixed bond to the iridal curtain, tying down the latter, so that light is entirely excluded at the peripheral side of the spot. But, as soon as the adhesion has given way, the liberated iris expands, the brown promontory becomes an islet, permitting light to pass freely around on every side; it, of necessity, offers far less obstruction than before, and vision is, in consequence, materially improved.

The question is not simply between the presence and

absence of these pigment spots, for no proof can be adduced of their more complete removal when the connected adhesions are not interfered with by atropine; on the contrary, we often meet with cases wherein old adhesions are skirted and covered by this colouring matter, which remains, apparently, quite as abundant as if, by timely interposition, the pupil had been set free. There is no ground, in short, for supposing that the continued attachment of the iris aids the process of absorption, or that its disconnection arrests that process. The question then, I repeat, is not between the presence and absence of pigment deposits on the capsule; but between the existence of such spots in a circumscribed isolated form,—and when prolongations from an adherent iris; in a word, between the continuance of such spots alone,—and their continuance plus iridal adhesions.

The foregoing remarks may be briefly summed up thus :

1. On plain anatomical grounds, cohesion of the iris and capsule is less to be apprehended; and, when happening, is less injurious, with an expanded than a contracted state of pupil.

2. The means of producing the former state should, therefore, always be employed in iritis; for though, when the complaint has reached a certain stage, the reaction of the pupil may be suspended; yet, in the majority of instances, this stage has not supervened when cases first apply.

3. Should the tentative use of belladonna not attain the desired object, its effect is for the time simply negative—is not injurious.<sup>1</sup>

4. Perseverance with belladonna or atropine, even when no immediate effect results, has this recommendation—that it takes advantage, at the earliest period, of the returning mobility of the iris, when inflammation abates.

5. During the convalescing stage, the detachment of adhesions is greatly promoted by the use of these agents, and, when accomplished, is a material and unmingled gain.

<sup>1</sup> Exceptional examples, wherein augmented pain follows the application, scarcely impair the universality of this rule; as the inconvenience rarely arises, and subsides on the discontinuance of the remedy.

# OPHTHALMIC CASES.

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BY JOHN F. FRANCE.

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CASES of artificial pupil brought to a successful termination, commonly, for obvious reasons, have an especial interest to the operator. Sometimes they may be supposed to possess a more general interest, either from the great extent of the previous injury, or some peculiarity in the condition of the organ, or in the means employed to restore its usefulness.

The subjoined examples occurring last year, illustrate, among other points worthy of consideration,—one, the great amount of injury, which, in a healthy subject, admits of remedy; the other, that the received opinion is not universally correct, that the protrusion of the iris in pouches, in connection with an occluded pupil, is produced by aqueous humour secreted in the posterior chamber and confined there. In this instance the so-called pouches remained, although the communication between the two aqueous chambers must have been fully re-established. The same case showed, in a striking manner, how small a pupillary aperture, if perfectly clear, is sufficient for the exercise of useful vision.

## 1. CASE OF ARTIFICIAL PUPIL BY EXCISION.

John M—, æt. 26, a Welshman, unable to speak English, was, on January 3d, 1855, injured by an explosion in a mine, near Aberystwith. His face was tattooed by the powder, and left eye quite destroyed. The right presented, on his admission into Guy's on September 1st, a large opaque cicatrix occupying the centre of the cornea, with which the entire pupillary margin was incorporated, so that vision, except of light and darkness,



was extinct. There remained, however, on the temporal and on the nasal side of this dense opacity, a transparent crescent, through which the iris was visible, apparently healthy in texture, stretched across the clear space, and separated from the lining of the cornea by a shallow vestige of anterior chamber.

The circumstances seeming favorable for the formation of an artificial pupil by incision (the crystalline having probably been evacuated through the wide corneal breach at the period of the injury), I introduced a cutting needle at the lower extremity of the nasal crescent, and directed its point to pierce and incise the iris at the upper part. Little inflammation ensued; but the aperture thus obtained proved insufficient for vision. On September 15th, having opened the lids with the spring speculum, I introduced the needle at the upper horn of the nasal crescent of transparent cornea, and passed it downwards, in the interval between the latter and the iris, which had apparently a greater depth of aqueous humour in front since the previous operation. By turning the needle's edge backwards, and retracting it once or twice with gentle pressure, I succeeded in opening a clear pupil sufficient to enable the patient, notwithstanding the escape of a little blood into the anterior chamber, to see to count his fingers, &c., on rising from the table. A little pain which he experienced in the course of the afternoon was relieved by cupping, and a pill of antimony with calomel and opium. The vascular excitement produced was inconsiderable; but in a day or two the new pupil, in spite of the application of atropine, contracted to a mere point, and vision was again extinguished.

I now therefore punctured the inferior portion of the cornea with Tyrrell's broad needle, inserted his hook at this opening, and that formed by the last operation in the iris, and thus drew out and removed a portion of the latter texture. Bleeding into the anterior chamber immediately obscured the effect; but no inflammation of consequence supervened; and as absorption of the blood proceeded, the newly formed pupil came into view, by means of which the patient could see to walk about, tell a gold from a silver coin, &c. There still, however, remained a filament of iridal structure crossing the pupil; and the space, though with this exception clear, was small. Hence, on



November 21st, I again pierced the upper boundary of the cornea with the broad needle, caught the upper edge of the artificial pupil on the hook, withdrew the iris, and removed a further portion with Maunoir's scissors, replacing the remainder *in situ* with a punctum probe. A pupil of ample dimensions and clear area was thus permanently established. Through this he enjoyed good vision for all ordinary purposes, and could distinguish large letters, &c. The poor fellow was very grateful; and on leaving hospital in December, expressed, in the broken English he had picked up, that he had rather possess the sight he had regained than all London. Amid the satisfaction attending his recovery of useful vision, he was not unmindful of the ornamental; and procured, at his own expense, an artificial eye for the left side, which fitted, worked, and looked well on the disorganized stump.

## 2. CASE OF ARTIFICIAL PUPIL BY EXTENSION.

A. B—, aged about 26, had cataract removed in boyhood successfully by Mr. Ware. Many years afterwards inflammation arose in the left eye, and the sight of that organ was lost. He therefore applied at a hospital, and an operation was determined on; but, according to the man's account, some difficulty being found in fixing the left eye, the surgeon suddenly resolved to operate on the right, the sight of which had up to that time remained pretty good. Probably some portion of opaque capsule was visible in the pupil, and a misunderstanding had existed from the first as to which eye it was proposed to operate on. Be this as it may, the result was unfortunate: inflammation supervened, and extinguished the sight of the previously useful globe. He applied, and was admitted at Guy's in the summer of 1855. The left eye was then hopelessly disorganized. The right presented a healthy conjunctiva, cornea, and sclerotic; but the iris was bulging forwards at the temporal side, and the pupil was contracted to a mere pin-hole occupied by false membrane, to which its margin was closely adherent. Vision was limited to the power of observing passing shadows.

I first operated with two needles through the cornea, in the mode suggested by Mr. Bowman, having the lids held apart with

the silver spring speculum. By these means the pupil was stretched open widely, and the aperture retained an ample size; but its enlargement only served to disclose a more extended surface of opaque membrane, which completely occupied the increased area. No inflammation of moment having resulted; after a suitable interval, I endeavoured, again with two needles, to tear through the remaining obstruction, but in vain; the capsule and false membrane receded before the needles, which thus could obtain no fixed point from which and on which to act.

I therefore shortly afterwards pierced the sclerotic with a Scarpa's needle, and transfixed the capsule, with the view of detaching it from the iris if possible, and depressing it beneath the axis of vision. The adhesions were, however, so strong, that steadily maintained pressure was inadequate to separate them, except by the exertion of an amount of force which would have endangered the normal ciliary connection of the iris. I succeeded notwithstanding in establishing a small opening between the edge of the pupil on the temporal side, and the expanse of white membrane which continued to occupy the greater portion of its area. The tension to which the texture of the iris was necessarily subjected, created great pain during this operation; but the inflammatory action subsided kindly in a few days. The artificial pupil, though of very limited size, then became so available that in September, three weeks after the last operation, the patient could recognise any ordinary object, and tell the time by the ward-clock without the aid of a glass. It was remarkable, that though the communication established between the two aqueous chambers through the new pupil must have been pretty free, yet the adjoining portion of iris, which bulged in pouches before the operation, continued to bulge afterwards. This would seem to indicate that the appearance was caused by interstitial deposit, rather than from confined aqueous humour, as commonly supposed.

September 15th.—With the assistance of a lens, the patient read easily the smaller type on his bed-ticket; he was shortly afterwards presented; and when seen some months subsequently, continued to enjoy satisfactory vision.

ON THE  
ALKALINE EMANATIONS  
FROM  
SEWERS AND CESSPOOLS.

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BY WILLIAM ODLING, M.B., F.C.S.

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IT seems that, of all atmospheric impurities, the emanations from decomposing excreta are most prejudicial to public health. The recondite chemical nature of these emanations is quite unknown. Hydro-sulphuric acid and sulphide of ammonium certainly exist, though I believe to a less extent than usually supposed, for I have not unfrequently met with specimens of sewer water which produced no sensible discoloration of lead paper. Moreover, if these sulphur compounds existed in much larger amount, we know that their toxic powers are incapable of effecting the morbid results observed.

At the suggestion of Mr. Simon, I have made a few preliminary experiments on the nature of sewer and cesspool exhalations.

The liquid contents of a cesspool, situated in Cross Street, Stockwell, were first examined. The construction of this cesspool is peculiar. It had been originally a shallow well, but latterly has served as a more perfect diverticulum for the excreta of the neighbourhood, by means of drain-pipes communicating with the surrounding houses. After subsidence, a portion of the tolerably clear liquid was evaporated to



dryness. An imperial gallon contained 95·36 grains of solid matter, including 26·28 grains of fixed organic matter. By gently warming another portion of the liquid, there was evolved a considerable amount of permanent gas, consisting principally of carbonic acid; but a little common air and a trace of hydro-sulphuric acid were also present. Half a gallon of the liquid was distilled until the distillate had no longer an alkaline reaction. It had a peculiar fetid ammoniacal smell. After saturation with hydrochloric acid, it was evaporated to dryness. The residue was dissolved in water, filtered, and precipitated with bichloride of platinum in the usual manner. The platinum salt weighed 58·80 grains, which, supposing it to be the ordinary platino-chloride of ammonium, would correspond to 8·95 grains of ammonia per gallon. The precipitate was then dissolved in water, and crystallized. The crystals were large and well defined. They occurred as flattened orange-coloured tablets, apparently modified octohedra, but certainly different from the ordinary crystals of platino-chloride of ammonium. A combustion of these crystals was made with chromate of lead. To the combustion tube was attached a U tube containing baryta water, so as to afford visible evidence of the presence of carbonic acid should any be produced. In the course of a few minutes, the baryta water became quite opaque, and so large was the amount of carbonic acid liberated, that the deposit of carbonate of baryta disappeared, owing to its conversion into bicarbonate. The further addition of baryta water reproduced the precipitate, which effervesced readily on the addition of hydrochloric acid. Thus it was rendered evident that the alkaline vapour from the cesspool was not simply ammoniacal, but carbo-ammoniacal.

Similar experiments were made with some liquid obtained from a sewer in Griffin Street, York Road, Lambeth. An imperial gallon of the liquid yielded 131·12 grains of solid matter, including 48·96 grains of fixed organic matter. Half a gallon was submitted to distillation. The first portions of the distillate were extremely pungent, and the alkaline reaction continued until nearly all the liquid had passed over. The smell of the distillate corresponded exactly with that of the other specimen. After saturation with hydrochloric acid, and evaporation to dryness, the alkali was precipitated with



bichloride of platinum. The platinum salt weighed 187·38 grains, which, supposing it to be the ordinary ammonio-chloride, would be equivalent to 28·56 grains of ammonia per gallon. The precipitate was dissolved in water and crystallized. The crystals were large, well defined, and altogether undistinguishable in their appearance from those previously obtained. A combustion of the crystals with chromate of lead was attempted, with the object of ascertaining the percentage of carbon; but, despite the introduction of a large quantity of copper turnings into the combustion tube, nitrous vapours made their appearance, so that the estimation was prevented. The chloride of calcium tube and the potash bulbs were then removed, and a U tube, containing baryta water, substituted. An abundant precipitate of carbonate of baryta took place.

A portion of the crystallized platinum salt was next incinerated. It gave 41·30 per cent. of platinum.

The Platino-chloride of Ammonia yields	.	.	44·36	} Per cent. of Platinum.
" " Methylamine "	.	.	41·64	
" " Ethylamine "	.	.	39·40	

Thus it appears probable that the crude volatile alkali of sewers contains a slightly larger amount of carbon than does methylamine, and a considerably less amount than does ethylamine. A second crop of crystals, obtained from the mother liquor of the preceding, gave 41·96 per cent. of platinum, and consequently contained a rather smaller proportion of carbon than did the original deposit.

It would be premature at present to hazard an opinion concerning the nature of the alkali. Despite the percentage analogy of composition, the crystals of the platinum salt obtained are unlike those of the platinum salt of methylamine. They bear a greater resemblance to those of the ethylamine platinic compound; and of course a mixture of about equal quantities of ammonia and ethylamine would correspond to the same percentage of platinum as methylamine. Should the alkali prove to be ethylamine, the subject would have a popular interest as suggesting the possibility of producing alcohol from sewer water. I am now distilling several gallons of

sewer water, and hope to obtain a sufficient quantity of the crude alkali to enable me to isolate and identify its carboniferous constituent. There is at present no evidence to show that this alkali is, or is not, the toxic ingredient of sewer exhalations.

ON THE  
DETECTION OF ANTIMONY  
FOR  
MEDICO-LEGAL PURPOSES.

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By WILLIAM ODLING, M.B., F.C.S.

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THE methods adopted by toxicologists for the detection of mineral poisons, after their reception into an animal organism, differ considerably in their general character from the processes used by chemists in ordinary analysis. This difference seems to be a necessary consequence of the peculiar circumstances attending medico-legal investigations. It rarely ever happens, that a chemist is called upon to identify the presence of a mineral substance, in a million times, or even several thousand times its weight of organic matter. Yet to such cases the efforts of toxicologists are constantly being directed. Moreover, the toxicologist very seldom meets with more than one mineral constituent in the same tissue, and consequently does not hesitate to employ methods, which, though admirable for the identification of a single metal, would somewhat impede the performance of an analysis professing to isolate and recognise every mineral ingredient that might possibly exist in the substance under examination. It is from this analytical point of view, that Fresenius objects to several of the most approved special methods of toxicological research, upon the grounds that they do "not contribute to the detection of other metallic poisons" than the one especially sought for, and that they contaminate the substance under investigation, with the metals used in the processes; but, as Dr. Taylor remarks, such "objections amount to

nothing in the hands of those who limit the application of the tests to the purposes for which alone they are intended."

The mode of recognising a metallic poison, most frequently adopted by the toxicologist, consists in a precipitation of the metal administered upon some other metal introduced by the analyst. Thus copper is deposited upon iron or platinum, mercury upon gold, arsenic upon copper, antimony upon copper or tin, &c. The metallic precipitate has then to be separated from the precipitant, and submitted to certain tests for its identification, which, moreover, is facilitated by an examination into the appearance of the deposit, and by a consideration of the circumstances under which it was formed. This plan of metallic precipitation differs from most other analytical methods, in its absolute independence of any influence arising from the presence of organic matter, and hence the preference usually accorded to it.

The subject of antimonial poisoning has recently acquired an unprecedented degree of importance. The symptoms produced by antimony, and the means for its detection, have been discussed with an interest little inferior to that excited by the major topic of strychnism. The late trial "*Regina v. Palmer*" was, I believe, the first criminal case on record, in which the metallic precipitation of antimony upon copper, according to the method of Reinsch, was employed for the separation and identification of the poison. Different opinions have been maintained with regard to the applicability of the process. In the works of Orfila and Christison, no mention is made of Reinsch's test, as a means for the recognition of antimony. In Dr. Taylor's work '*On Poisons*,' it is simply remarked, that "muriatic acid and copper will also serve as a useful trial test." In the last edition of his '*Medical Jurisprudence*,' we find it stated, after a reference to Marsh's test, that "Reinsch's process also serves to separate antimony from the soft parts, when they are boiled for some time with muriatic acid and water. The copper acquires a violet-blue or steel-gray colour (resembling arsenic), according to the proportion present." No directions are given for the subsequent examination of the deposit.

I purpose in this paper to consider the merits of Reinsch's process, as used for the detection of antimony, and to give the



results of some experiments made with the view of ascertaining, firstly, whether any other metals are capable of affording similar deposits under similar circumstances, and secondly, what means are best adapted for the identification of the antimonial deposit.

*Delicacy of the test.*—When  $\frac{1}{1000}$ th of a grain of tartar emetic—that is, exclusive of the water of crystallization—is dissolved in 100 grains of dilute hydrochloric acid, and a piece of clean copper foil exposing one square inch of surface introduced, after five minutes' ebullition, there was a well-marked discoloration; and after a quarter of an hour, a decided steel-coloured deposit. The ebullition was performed in a small flask, to which a long tube was attached, so that the original bulk of the liquid was preserved by the reflux of the volatilized portions. The experiment was repeated with a piece of copper foil exposing two square inches of surface, and in this case, after three quarters of an hour's ebullition, there was produced only a very delicate deposit, quite insufficient to conceal the red colour of the copper. These results correspond generally with those afforded by arsenic. The  $\frac{1}{1000}$ th of a grain of arsenious acid will deposit upon two square inches of copper surface a decided coating, although, indeed, the colour of the copper is not altogether overcome. But arsenious acid contains 75.6 per cent., and dry tartar emetic only 39 per cent. of metal. It seems, therefore, that with equal weights of each metal, equal effects would be produced; but with arsenious acid and tartar emetic respectively,  $\frac{1}{2000}$ th of a grain to an inch of copper surface is the practical limit of the former, and  $\frac{1}{1000}$ th of a grain to the same amount of surface the limit of the latter. In depositing arsenic upon copper, the proportion of hydrochloric acid may be increased, if not with positive advantage, at any rate without injury, to the extent of 50 per cent. by volume; but in the case of antimony, an increase above 20 per cent. appears to interfere with the delicacy of the test. The colour of the antimonial deposit resembles very closely that of the arsenical. It has, however, more of a blue or violet tint.

*Effect of dilution.*—I find that when  $\frac{1}{1000}$ th of a grain of emetic tartar is dissolved in 500 grains of dilute hydrochloric acid, and a square inch of copper surface introduced, after half

an hour's ebullition, a decided and characteristic deposit is produced; but with 1000 grains of liquid, even after an hour's ebullition, there was only a faint discoloration. A dilution therefore of 500,000 times constitutes the practical limit to the application of the test; whereas with arsenious acid, a very excellent deposit may be obtained from the  $\frac{1}{1000}$ th of a grain, dissolved in 2000 grains of liquid, that is, with a dilution of 2,000,000 times. The delicacy of Reinsch's process, as a means for evidencing the presence of antimony, is much greater, however, than that by sulphuretted hydrogen. One hundred grains of a solution containing the  $\frac{1}{1000}$ th of a grain of tartar emetic becomes faintly, almost imperceptibly, coloured by sulphuretted hydrogen, and, after some hours' subsidence, a very slight orange-coloured precipitate is manifested; but with the same amount of emetic, and 500 grains of fluid, sulphuretted hydrogen gives no indication whatever of the presence of the metal. The deposition of antimony upon copper takes place, though incompletely, at ordinary temperatures; but for the deposition of arsenic, even when existing in large quantities, ebullition, or at any rate a temperature approaching that of ebullition, is indispensable. With a solution containing 1.0 per cent. of tartar emetic, the precipitation upon copper in the cold takes place somewhat rapidly. When the antimonial deposit, produced by boiling, is slight, it adheres firmly to the copper, but otherwise it is apt to rub off. Hence, it is advisable not to use filtering paper for the purpose of drying the foil or gauze, but simply to apply a gentle heat, that of a water bath for instance. The deposit when dry has very little tendency to rub.

*Precipitation of other metals.*—I can add nothing to what is generally known concerning the precipitation of arsenic and mercury upon copper; but shall remark upon the possibility of precipitating bismuth, tin, silver, platinum, lead, cadmium, and zinc, so as to simulate the antimonial deposit.

*Bismuth* is readily precipitated from its acidified solutions, even at ordinary temperatures, by the immersion of clean copper. With a solution containing 0.1 per cent. of terchloride of bismuth, the precipitation in the cold commences almost immediately after the introduction of the copper. With a solution containing 0.01 per cent., and at the temperature of

ebullition, the precipitation of bismuth takes place in a manner resembling that of antimony under similar circumstances. When the deposit of bismuth is thin enough to allow the copper to show through, the appearance presented by the foil approximates sufficiently to that produced by antimony to mislead an unpractised observer; but, as the deposit of bismuth increases, the resemblance diminishes. The metal is ultimately deposited in a very brilliant condition, and of a whitish colour. Like arsenic, it adheres firmly to the copper. Its tint is intermediate between that of a mercurial and that of an arsenical deposit. The  $\frac{1}{1000}$ th of a grain of terchloride of bismuth will not give a perfect coating to one square inch of copper surface. A dilution of 100,000 appears to form the limit of the test, which, as applied to bismuth, is consequently less delicate than is the sulphuretted hydrogen test.

*Tin.*—The results obtained with tin are somewhat anomalous. Certain irregularities are presented, for which, at present, I am unable to account. Bichloride of tin yields a deposit with *extrême* difficulty, and only after protracted ebullition. With protochloride of tin, I have not succeeded in obtaining any incrustation, when the dilution exceeded 500 times. With that degree of dilution, I have sometimes obtained slight, though obvious deposits. On the other hand, I have frequently failed to obtain any discoloration with a dilution of only 100 times. The amount of hydrochloric acid certainly affects the result. The best proportion that I have found is about 20 per cent. A much greater or less quantity interferes considerably with the action of the test. With solutions, however, containing 5 per cent. and upwards of protochloride of tin, there is no difficulty. The appearance of the stannic incrustation is liable to great variations. Sometimes it is of a dark, almost black colour; but at others it approximates very closely to the steel-blue colour of the antimonial deposit, than which, indeed, it is somewhat less brilliant and much less uniform. When the coating of tin is thin, it has a peculiar dotted look. Upon heating the coated foil in a water bath, the deposit sometimes undergoes a great alteration in its appearance, and seems to diminish considerably. Prior to the drying of the foil, the coating of tin is very readily removed, even by a slight friction. I fancy this non-adhesion is



increased when the deposit is obtained from an organic liquid, in which case also there is usually a greater want of brilliancy.

*Silver* is generally precipitated in the pulverulent state on the introduction of copper into its solution, the deposition being independent of temperature. But a brilliant, uniform, argentine coating may be produced upon copper, by making a very dilute solution of sulphate of silver slightly acid with sulphuric acid, and then immersing the foil. Almost equally good results may be obtained with chloride of silver, when it is in that finely divided non-subsiding condition in which it occurs when precipitated from cold dilute solutions by an excess of hydrochloric acid. The colour of the argentine coating is sometimes white, but more often of a dark colour, such as is produced occasionally when the experiment of reduction by aldehyd is badly performed. In this case, the silver deposit might possibly, though not probably, be mistaken for that of antimony. The presence of nitric acid interferes with the brilliancy of the coating.

*Platinum*.—When copper foil is immersed in a very dilute cold solution of bichloride of platinum, it speedily acquires a shining metallic coating, similar to that produced by bismuth; but on removing the coated foil, and exposing it to the air, the colour changes to a steel-blue, very closely resembling that of some forms of antimonial deposit.

*Lead*.—With a concentrated solution of chloride of lead, acidulated with hydrochloric acid, and more especially in the presence of organic matter, it is possible by prolonged ebullition to obtain a deposit upon copper resembling somewhat that produced by antimony. The deposit is, however, extremely slight, and devoid of any blue or violet colour.

*Cadmium* affords results of considerable interest. When metallic cadmium is introduced into an acidulated or non-acidulated solution of chloride of copper, with, or more slowly without, ebullition, the whole of the copper is deposited in a red pulverulent state. The liquid after filtration does not become discoloured by excess of ammonia; and the precipitate produced by ferrocyanide of potassium has not the slightest tinge of red or brown. But when a piece of clean copper foil is immersed in a boiling, moderately concentrated, and slightly



acidulated solution of chloride of cadmium, after some little time a very brilliant coating of cadmium, of a white or yellowish-white colour, is precipitated upon the copper. These antagonistic results must, I suppose, be referred to the superior action of superior masses.

*Zinc* solutions, according to Reinsch, are capable of imparting a deposit or tarnish to copper. I have not, however, succeeded in obtaining such a result, though I have tried the experiment, variously modified, on several occasions.

From the above experiments, I conclude, firstly, that deposits of arsenic and antimony most closely resemble each other. The resemblance is, however, of no practical consequence, owing to the extreme ease with which the arsenical deposit is identified. Secondly, that under the same conditions of experimenting employed for the deposition of antimony, bismuth, and even tin, may yield metallic deposits; and that in a criminal case it would not be safe to rely upon the appearance of the antimonial coating, as a means of distinguishing it from either of the other two. At the same time, the appearance of the bismuth deposit, as I have seen it, is somewhat characteristic, and a deposit of tin would only occur when the metal existed in such large quantities as to present no difficulty of detection. Thirdly, that it is just possible for a deposit of lead or platinum to be mistaken for one of antimony. Moreover, I think it not unlikely that in very dilute solutions of silver and mercury no obvious precipitation might take place until the temperature of the liquid was raised; and in this case, each of the two metals could originate a fallacy. Fourthly, that zinc is not precipitable upon copper, and that cadmium is not precipitable under the same conditions as antimony, that is to say, in the presence of much hydrochloric acid.

*Examination of the antimonial deposit.*—Seeing then, that the production and appearance of the metallic deposit are insufficient to demonstrate its antimonial character, what steps are we to take for its identification. With regard to arsenic, as I have already observed, the means for effecting its separation from the copper foil or gauze, by sublimation and oxidation, are so delicate, so facile, and so characteristic,

as to leave nothing further to be wished for. A ready method, however, of isolating antimony from the copper on which it is deposited has hitherto been a desideratum, and the want of such a method has no doubt militated against the general reception of Reinsch's process as applicable for the detection of antimony. It would of course be quite practicable to dissolve the two metals in acid, and then to separate the copper from the solution by the ordinary reagent, namely, sulphide of potassium. But this process, besides being troublesome and inelegant, affords very unsatisfactory results. Nitro-hydrochloric acid is required to effect the solution of the metals. The residual nitric acid has to be expelled by an evaporation to dryness. Very much sulphide of potassium must be used for the effectual precipitation of the copper, and then, on the acidulation of the filtrate, what with the nitric acid, and what with the necessarily large amount of sulphide of potassium employed, a pale precipitate is produced, in which it is quite impossible to recognise the orange-coloured antimonial sulphide, unless, indeed, the antimony existed in very large proportion.

What we require is, some means for dissolving metallic antimony, having no solvent action upon metallic copper. The ingenious method adopted by Dr. Taylor for effecting this separation is described in his own words, as follows: "We cut up some of the gauze containing the deposit, we heated it with nitrate of soda in a platinum crucible; that converted the antimony into antimoniate of soda; that was dissolved, or rather diffused, in water containing a little hydrochloric acid, and precipitated by sulphuretted hydrogen. The precipitate after twenty-four hours was deposited; it had a reddish-brown colour, like the sulphuret of antimony, and, like it, was soluble in strong hydrochloric acid."<sup>1</sup> I have found, however, on repeating this process, that although, when employed cautiously, and in cases where the amount of deposit is moderately large, it yields satisfactory results, yet that very frequently, and always when mere traces of the poison are present, the precipitate finally produced presents an appearance anything but characteristic of antimony. There is always a certain contamination with the sulphides of copper or platinum, which

<sup>1</sup> This test was not relied upon exclusively.

accounts for the reddish-brown colour of the precipitate, as mentioned by Dr. Taylor.

Dr. Hofmann pointed out to me, some time back, the property possessed by tartaric acid, of dissolving antimony from the silver compound  $\text{Ag}^3\text{Sb}$ , precipitated by the reaction of antimoniucretted hydrogen upon nitrate of silver. That tartaric acid, and cream of tartar likewise, do effect the decomposition of this silver compound, and the solution of the antimony, is undeniable; but neither of these agents exerts any sensibly solvent action upon metallic antimony precipitated on copper, the difference being probably due to the different state of aggregation in which the metal exists. Among the distinctive properties of the metals, copper and antimony, it occurred to me, that one of the two following might be made available for their separation, namely, the volatility of chloride of antimony, and the solubility of oxidized antimony in alkaline solutions. From a few experiments made by passing chlorine gas over the coated copper, I have no doubt as to the feasibility of the first-mentioned plan, which, however, owing to the very successful results obtained by the other process, I did not pursue into detail.

In reference to this other process, after experimenting with several reagents, according to plans variously modified, I found the following method to answer admirably. A solution of permanganate of potash was made of such a strength, as to have the colour of ordinary red cabbage infusion (about one grain of the crystallized salt to fifteen ounces of water). The coated copper was introduced into a test tube, and covered with this solution. A drop or so of solution of potash was then added, and the whole boiled. In the course of a minute or two, the colour of the permanganate became completely destroyed, with the formation of a precipitate of red oxide of manganese. The liquid was then filtered, acidulated with hydrochloric acid, and treated with sulphuretted hydrogen, which immediately produced the characteristic precipitate of antimonial sulphide, of a pure orange colour. The advantages of this process consist, not only in the excellence of the result, but also in the facility and rapidity of its application. One ebullition, one filtration, and one reaction, are all that is required for the complete identification of the deposit. But



should it be deemed desirable, additional tests may be applied to the precipitate, even when obtained in most minute quantity. Thus, I took 500 grains, by measure, of a hydrochloric decoction of a stomach, added to it the 0·001 of a grain of tartar emetic, and boiled, in the liquid, a piece of copper gauze for three-quarters of an hour, by which time it had acquired a decided opaque coating. It was then washed and treated with a little of the permanganate solution, as above described. The addition of sulphuretted hydrogen to the filtered and acidulated solution produced an exceedingly slight yellow discoloration; and by the next morning, there was an evident orange coloured precipitate. The supernatant liquid was carefully poured off, and a drop of ammonia added, when the precipitate disappeared. A drop of hydrochloric acid reproduced the orange turbidity. One or two more drops of acid were added, and the whole boiled, when the turbidity disappeared. Into this clear liquid was introduced a small piece of tin-foil, which, in the course of half an hour, received a decided black discoloration. Thus is it possible to detect with certainty, by a succession of tests applied to the same portion of metal, the 0·001th of a grain of emetic tartar, dissolved in half a million times its weight of an organic liquid.

In effecting the solution of the antimonial deposit by means of permanganate of potash, only a drop or so of potash solution should be added, for if too much be used, the red colour of the permanganate disappears upon heating, but is replaced by the green colour of the manganate, which salt requires several minutes' ebullition for its complete decomposition. By using a very small proportion of potash, moreover, the ordinary *Liquor Potassæ* may be employed, the trace of lead usually contained therein not affecting the result.

Of course before using this process, it is advisable to heat the coated foil in a subliming tube, so as to negative the presence of arsenic. Arsenical deposits do not yield good results with the permanganate liquid, inasmuch as a portion of the arsenic remains combined with the precipitated red oxide of manganese, and that portion which is dissolved is in the state of arsenic acid, and consequently difficult to precipitate with sulphuretted hydrogen. Tin deposits, moreover, are not readily dissolved by the permanganate liquid, though when the deposit



is considerable, a faint yellow precipitate may be obtained by the action of sulphuretted hydrogen on the filtered and acidulated liquid. Bismuth deposits, by similar treatment, do not afford a trace of metal in the filtered solution.

The copper-foil, whether coated with arsenic, antimony, bismuth, or tin, presents, after the action of the permanganate solution, different appearances, according to circumstances. Occasionally it assumes its red colour by simple ebullition with the permanganate; generally, it has a dark, dull colour, from the formation of oxide, until hydrochloric acid is added, which brings out the characteristic appearance of clean copper; while, sometimes, the addition of the acid reproduces the normal appearance of coated copper, showing that a part only of the original deposit had been acted upon by the permanganate liquid. These differences obviously depend upon the amount of deposit, the proportion of permanganate, and the nature of the metal.

In addition to antimony, tin-foil is capable of precipitating the metals nickel, cobalt, cadmium, lead, bismuth, copper, silver, mercury, arsenic, platinum, and gold, from their solutions; but under the circumstances in which it was employed, in the experiment described a few paragraphs back, the discoloration could only be due to antimony. The plan of precipitating antimony upon tin has much the same delicacy as the sulphuretted hydrogen test, a dilution of 100,000 times forming the limit of the reaction; but, when the antimony existing in a large amount of liquid is first concentrated upon copper gauze, and then dissolved in the smallest possible quantity of liquid, both the sulphuretted hydrogen and the tin tests become exceedingly sensitive.

ON THE  
GASTRIC JUICE  
AS A  
SOLVENT OF THE TISSUES OF LIVING ANIMALS.

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By F. W. PAVY, M.D.

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It is now one of the most accredited facts in physiology that the glandular follicles belonging to the walls of the stomach, secrete, upon the application of the stimulus of food, an acid fluid containing a peculiar animal principle—pepsine—which, at the temperature of the living body, acts as a powerful solvent of the azotized constituents of animal and vegetable substances, reducing them to a fluid condition, in which they can be subsequently absorbed in their transit along the alimentary canal; and, after entering the circulatory system, can be transmitted throughout the animal economy, to be rendered subservient to the processes of nutrition.

The ingenious experiments on the subject of digestion, that were performed with a zeal and perseverance but rarely observed, towards the latter part of the eighteenth century, by the Abbé Spallanzani, cleared up many points that were previously only resting upon conjecture. He showed that the gastric juice exuded through pores situated upon the inner surface of the stomach, and proved that it was in this viscus, and by means of this fluid, that the solution of food was effected. Enclosing different kinds of alimentary materials in linen bags and perforated metal tubes, he forced them into the stomachs of animals, and even swallowed them himself, to observe the action that took place. And, by means of sponges similarly introduced into the stomach and afterwards withdrawn, he was enabled, from time to time, to collect a considerable quantity

of gastric juice and to perform experiments upon the artificial digestion of food. In those dark ages of organic chemistry, however, belonging to his times, the constitution of the gastric juice was most imperfectly determined; and the author in question was even led to deny its acidity, except during the digestion of *certain* materials, which he thought communicated to it its then acid character. Spallanzani, indeed, failed to recognise the difference in the quality of the secretion of the stomach, during its opposite conditions of repose and functional activity.

It was in the year 1822 that a most remarkable opportunity occurred of witnessing the process of digestion whilst being carried forward in the living stomach of a human subject. Alexis St. Martin, a young Canadian, accidentally received from a musket, but a yard distant from him, a charge of duck-shot, which penetrated his left side and laid open the abdominal and thoracic cavities. He made a most miraculous recovery, the wound healing with the exception of a fistulous opening communicating with the interior of the stomach, which ever afterwards remained. Through this opening, the nature of the operations going forward during digestion could be viewed, and materials abstracted or introduced at pleasure. Being at different periods until 1833 under the charge of Dr. Beaumont, several series of interesting and well-devised experiments were performed, which have done much to advance our knowledge and to give us accurate data upon the subject of digestion.

Since these experiments on St. Martin, fistulous openings have been established in the stomachs of the lower animals, and I have now in my possession a dog in this condition, that has served me as subject for the succeeding experiments on the digestion of the tissues of living animals; some of which experiments, it is due to remark, are but repetitions of what have been performed by others.

The dog in question is a most quiet and affectionate little creature, belonging to the terrier breed. Her portrait has been given by our artist in the accompanying plate. It well represents the point of interest; but is far from being a flattering likeness, for she has naturally a sharp and lively appearance. The opening is seen at the upper part and to the left side of



the abdomen, and projecting through it is one end of the silver canula figured entire in the lower left-hand corner of the plate. The other end of the canula is situated in the interior of the stomach, which is, therefore, in immediate communication with the exterior of the body. The canula is constructed of two pieces, the one screwing into the other, so that its length can be increased or diminished, according as required by the thickness of the abdominal parietes of the animal. Its bore is a little more than half an inch in diameter; and, by means of a cork, the contents of the stomach are easily prevented from escaping. The operation of making the fistula was performed in the early part of April, 1855, whilst the animal was under the influence of chloroform. An incision being made through the abdominal parietes, the anterior surface of the stomach was exposed; and, by means of a small aperture through the walls of this viscus, one end of the canula was inserted into its interior. The body of the instrument being subsequently placed in the incision through the parietes of the abdomen, the edges of the wound on each side were adjusted and closed by sutures around it, leaving it firmly fixed with its other end projecting externally, in which position it has remained ever since. To prevent the stomach from escaping or slipping from the canula, the former was secured by a couple of sutures to the abdominal parietes until after adhesion had taken place, when the sutures were removed. The animal manifested very little disturbance of health, and in the course of a week appeared quite recovered. Notwithstanding it is nearly eighteen months ago that the fistula was made, yet she is now in as good a condition and as lively as ever she was. Being pretty well used to my experiments, she will lay, without attempting to move, for two or three hours at a time, with a frog's hind legs in her stomach, whilst its tissues are being digested.

The power of the stomach of digesting living substances, if undoubtedly established, as we shall presently find it to be, is not only a fact of physiological interest, but likewise of importance, as standing in direct antagonism to the doctrines that have been handed down to us from the illustrious Hunter, with regard to the *post-mortem* solution of the stomach, which in some cases is observed to take place.



In the 'Philosophical Transactions' for 1772 is a communication, which was read before the Royal Society, June 18th of that year, by John Hunter, "On the Digestion of the Stomach after Death." He notices that, in occasional instances, especially in persons who have died of sudden and violent deaths, on making an inspection of the body, the great extremity of the stomach is found so dissolved as to have allowed of the escape of its contents into the abdominal cavity. He also states that, where even a perforation of the stomach may not be observed, yet there are very few dead bodies in which the stomach is not at its greater end in some degree digested; this partial digestion being indicated by a thinness and transparency of the membrane, and by the blood which the vessels contain passing out at their digested ends when pressure is made from the larger towards the smaller branches, and appearing like drops on the inner surface.

Having observed the greatest extent of solution in cases where the individual had been in the enjoyment of perfect health up to the moment of death, he very naturally inferred that such a phenomenon could not be the result of disease in the living subject, but must be owing to the action of the digestive fluid after death had taken place. If, then, the mucous lining of the stomach secreted a fluid which was capable after death of digesting the viscus itself—if, in fact, the walls of the stomach were not constructed of indigestible materials; an important question arose, to determine what protected the organ during life from undergoing the same solution as the food received into its cavity. Hunter referred it to the *living principle*. To use his words: "The stomach which at one instant, that is, while possessed of the living principle, was capable of resisting the digestive powers which it contained, the next moment, viz., when deprived of the living principle, is itself capable of being digested, either by the digestive powers of other stomachs, or by the remains of that power which it had of digesting other things." In illustration of his views he also asserts, "If it were possible for a man's hand to be introduced into the stomach of a living animal, and kept there for some considerable time, it would be found that the dissolvent powers of the stomach could have no effect upon it; but if the same hand were separated from the body, and

introduced into the same stomach, we should then find that the stomach would immediately act upon it."

It is clear from these doctrines, that the living principle, in combination with a substance, is supposed to be capable of protecting it from the action of the digestive process; and that the stomach, during life, thus acquires its immunity from undergoing the same solution as the food introduced into its cavity. Before proceeding further, let us see if this theory be supported by fact; if, indeed, the living principle possess the power attributed to it. In a dog with a fistulous opening in its stomach, the experiment is readily performed. It is easy to introduce through the instrument some member of a living animal, and note the result that takes place.

If our dog be fed, so as to call the stomach into functional activity, and excite the secretion of gastric juice (for it is only during the presence of food or some foreign substance in the stomach that gastric juice is secreted, the inner surface of the organ being at other times moistened with a fluid of a bland mucous character), and about two hours afterwards, the hind legs of a living frog be introduced through the canula, and be fixed or held in that position; the process of digestion will commence, and proceed until the whole of the skin, flesh, and bones of the extremities projecting in the stomach have been completely dissolved, the frog remaining all the while alive, and even continuing so for many hours afterwards. If the animal be removed after its extremities have been digesting about two hours and a half, they will present the appearance represented in the sketch on the accompanying plate, which has been drawn by our artist from nature.

This experiment has been oftentimes performed on the Continent, and I have several times repeated it myself, with always a similar result, so that there cannot possibly exist any question with regard to the power of the stomach of dissolving living substances. But it can scarcely be adduced as a conclusive argument against the living principle protecting the stomach from being digested by its own secretion; for it might be urged that the living principle in the frog—a reptilian and cold-blooded animal—is so weak as compared with a warm-blooded quadruped, that the gastric juice of the latter is capable of overcoming it, whilst it might be incapable

of acting upon the living tissues of the higher animals belonging to its own class. To remove all objection on this point, I therefore performed the following experiment with the ear of a living rabbit.

In November, 1855, three hours after my dog had been fed on its usual daily meal (a bundle of tripe), the ear of a full-grown healthy rabbit was carefully inserted through the canula into its stomach, some pieces of tripe having been previously removed to make room for it. The dog lay perfectly quiet on her side during the whole of the experiment, and the rabbit was held in the necessary position with the hands, so as to avoid in any way obstructing the circulation through or mechanically injuring the ear. At the end of two hours' time it was withdrawn, and several spots of erosion, some as large as a sixpenny-piece, were observed on its surface, but nowhere was it eaten completely through. On being replaced for another two hours and a half, the tip which projected in the stomach was almost completely dissolved, a small remnant of it only, being left attached by a narrow shred to the remainder of the ear. The gastric juice seemed to act like a strongly corrosive material, making first a number of ulcer-looking spots through the skin, and afterwards extending its action more rapidly through the central parts of the ear. A rather profuse hemorrhage took place, especially during the latter part of the process of gastric solution, and the rabbit at times manifested considerable disturbance at what was going forward. The sketch placed by the side of the frog was made on the day following the experiment, and forms a good representation of the appearance the digested ear presented. Three days after the experiment, the rabbit refused to eat its food as usual. The base of the ear was exceedingly hot and œdematous, and remained so till the fifth day, when the animal died, apparently of erysipelas or phlebitis, for there was considerable purulent infiltration of the parts. I have preserved its head as a dry preparation.

This experiment, I think, may be looked upon as a conclusive proof, not only that the *living principle*, or, as we now term it in our more modern phraseology, the *vital force*, is incapable of counteracting the solvent action of the gastric juice, but likewise insufficient to explain the exemption of the stomach during life from being destroyed by digestion. The



tissues of the rabbit's ear are combined with the same living principle as the walls of the stomach, and the former have even the further advantage of being naturally of a more indigestible character. I have not the slightest doubt in my mind, that had I have been disposed to have introduced my own finger into the dog's stomach instead of the rabbit's ear, it would have similarly undergone digestion. Indeed, my fingers, during the experiment, becoming moistened with the gastric juice which escaped from the stomach, afterwards felt quite tender, as if the cuticle had been partially removed.

If, then, the vital force, which physiologists are too apt to fall back upon, and assign as a cause of phenomena, which appear otherwise unintelligible; if, I say, this vital force be brought forward upon grounds that in this case can be negatived by direct experiment, we thus release ourselves from such a fetter, and the reason the stomach escapes digestion during life, and undergoes solution after death, again becomes an open question. The following explanation has been advanced by M. Bernard, of Paris, which is at the same time ingenious, and, I believe, consistent with truth.

The stomach is lined with an epithelial layer that secretes a mucus, and acting as a varnish, protects the deeper tissues. During life, the epithelium and mucus are constantly being reproduced, which is not the case after death. When food is introduced into the stomach, gastric juice is poured out and fulfils its physiological intention. Coming in contact with the inner surface of the stomach, it also acts upon the mucus and epithelium, and leads to their solution; but whilst this is taking place, a fresh supply is produced, which still continues to protect the viscus until the process of digestion is complete, when the organ has a period of repose. Under this view, the stomach is no more exempt from the action of the gastric juice than the food introduced into its cavity, but the reproduction of epithelium and mucus prevents the solution or digestion of the deeper parts, by preserving them from exposure to contact. When death has taken place, the epithelial layer is no longer reproduced, the gastric juice acting upon it, arrives in contact with the subjacent structures, and causing their solution, leads to a perforation of the parietes of the organ, varying in extent according to the amount of digestive fluid present.



From this physiological consideration, we have a plausible explanation of the pathology of that exceedingly rare affection—perforation of the stomach, where the symptoms appear suddenly during an apparent state of health. In such cases, from a deficient tone in the system, the production of epithelium and mucus may not be sufficiently abundant for the security of the organ. The gastric juice unawares arriving in contact with the deeper coats, acts upon them, and produces a perforation that suddenly allows of the escape of food into the abdominal cavity.

The reason that solution of the stomach after death is observed in one case and not in another, is most easily explained. In by far the generality of subjects examined on the post-mortem table, there is an absence of perforation, and at most a softening and thinning of the greater end of the stomach. In such instances there was little or no gastric juice in the stomach at the period of death, as will be the case in the majority of deaths taking place from natural causes. If the life of a person, however, be suddenly removed whilst the stomach is charged with food that is undergoing the process of digestion, there will then be gastric juice sufficient to act upon and perforate the coats of the viscus, and allow of the escape of its contents into the peritoneal cavity. From the nature of the conditions requisite for the production of this phenomenon, it is but rarely observed in the human subject. Those, however, who have been accustomed to the dissection of the bodies of the lower animals, suddenly sacrificed for experiment or otherwise, must have constantly met with its occurrence.

### PLATE VIII.

ILLUSTRATING DR. PAVY'S EXPERIMENTS ON THE ACTION OF  
THE GASTRIC JUICE ON LIVING ANIMALS.

The lower figure represents the dog, with the fistulous opening in its stomach; and at the side is seen the canula, which is inserted into it.

The figures of the rabbit and the frog exhibit the solvent power of the gastric juice on the ear and legs of these animals, respectively.







ON  
POISONING BY STRYCHNIA,  
WITH  
COMMENTS ON THE MEDICAL EVIDENCE GIVEN AT  
THE TRIAL OF WILLIAM PALMER.

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BY ALFRED S. TAYLOR, M.D. F.R.S.

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THE attention of the profession and public has been of late especially directed to the subject of poisoning by STRYCHNIA, from the facts and opinions elicited on the trial of WILLIAM PALMER, for the murder of JOHN PARSONS COOK. This trial, which occupied the Central Criminal Court for the unusually long period of twelve days (14th to 27th May, 1856), will be for ever remarkable in the annals of jurisprudence and legal medicine. An Act of Parliament was expressly passed for the purpose of moving the proceedings from the county of Stafford, in which the crime was alleged to have been committed, to the metropolis, in order that the accused might have the benefit of an impartial jury. Three learned Judges, the Lord Chief-Justice Campbell, Mr. Baron Alderson, and Mr. Justice Cresswell—men eminent for their knowledge of criminal law—presided on this occasion. The prosecution was conducted with consummate ability, as well as moderation, by the Attorney-General, assisted by four distinguished members of the bar; and among the numerous witnesses for the prosecution and defence, were men whose names are well known to science, and many of whom occupy a high professional position in London, Dublin, and Edinburgh. In spite of the most strenuous efforts made to “save the life of the prisoner,” by

the display of an unusual amount of sophistry, by a misrepresentation of medical facts and opinions, by personal attacks on witnesses whose evidence was of vital importance in the case—the last refuge of a failing defence—and finally, by an attempted intimidation of the jury, the prisoner was convicted, and has since suffered the punishment justly due to his crime.

If we seek for an explanation of the extraordinary excitement produced in the public mind by the facts elicited at the inquest on the body of JOHN PARSONS COOK, and at the trial of WILLIAM PALMER, we shall probably find it, so far as this crime is concerned, in the deadly and insidious nature of the poison selected,—the name of which was until then hardly known to the public,—in the ingenious mode of administration, *i. e.*, by the substitution, on two successive nights, of poisoned pills for pills prescribed by a medical man; and in the fact, that the accused was himself a member of the medical profession, a man of education and knowledge, as well as of a certain degree of respectability. The sudden deaths which had taken place in his house during a period of four years—of three children, of a friend while on a visit, of his wife's mother, and lastly, of his wife, for whose murder by poison he was to be immediately tried in the event of an acquittal on the charge which he was then called upon to answer—gave an interest to these proceedings far beyond that attached to an ordinary case of poisoning. With a prevalent conviction of the prisoner's guilt, there was, at the same time, an almost universal feeling that his trial for the murder of Cook would end in an acquittal. His legal advisers felt confident of a verdict in his favour. Whence arose this feeling? The crime had been committed in secrecy, with consummate skill and art; and the poison to which death was ascribed by the witnesses for the crown, was not found in the body of Cook. The case, therefore, had to be made out to the satisfaction of the jury upon other than chemical grounds; and this was thought to be a difficulty which the skill and experience of the legal advisers of the crown would never surmount, although aided by the advice and opinions of many medical men whose names and reputations were well known to the public, who could have no object in coming forward to give evidence on the occasion, than that of upholding the principles of truth and justice.

It was plausibly stated, and widely circulated by a portion of the press, before and pending this trial, that no man can die of poison except poison be found in his body, and that unless the matèrial instrument of death be *always*, and *under all circumstances*, forthcoming, upon such charges, no man's life would be safe! The varied aspects of disease were described as such that no one could, under any circumstances, trust to symptoms and appearances as evidence of death from poison. The other side of the picture was studiously concealed from the public view; namely, that to men of craft and skill in the medical profession, deadly poisons are accessible, which may destroy life in such doses and under such modes of administration, that, while no chemical tests can reveal their presence in the body, their unlawful use may be surely and satisfactorily indicated by the suddenness, intensity, and peculiarity, as well as the fatal rapidity of the symptoms which they produce. There are other poisons accessible to professional men, which, according to the mode of administration, may be detected under one set of circumstances, while the tests will wholly fail to detect them in other cases. It was studiously concealed that, while the chemist's art is utterly valueless to aid the law in cases of this description, physiology and pathology will enable the medical observer to detect the crime. If the poison be administered, by whatever channel it may enter the body, there will be the symptoms to announce its effects. Due caution is of course required in drawing inferences from symptoms, but an equal, if not a greater amount of caution, is also demanded in drawing inferences from the results of delicate and refined chemical processes applied to the solids and fluids of the dead body. Is the chemist more certain of the accuracy of the tests employed in such cases, than the experienced physician of the symptoms? Take an instance in which the symptoms are so doubtful that they might be assigned to strychnia or disease. The chemist demonstrates, as he says, by certain colours, the presence in the dead body of the fifty thousandth or the twenty thousandth of a grain of poison; one of a sanguine temperament will tell you that, beyond all doubt, it is strychnia; a second will affirm that the appearance is equivocal; and a third will tell you that he disbelieves altogether that it indicates the presence of



the poison. This, as it will be seen hereafter, was very much the state of things in the case of Cook; and it is not at all improbable, from the kind of evidence given at the trial, and the bold reliance placed upon infinitesimal results, that had the chemists for the defence changed places with the chemists for the prosecution, the prisoner would have been chemically convicted by his own witnesses. Is it upon a slender reed like this that the public are to be taught to lean to protect themselves from death by poison? Let it be remembered, that if the physician, as a pathologist or physiologist, may be deceived by symptoms, the chemist may be equally deceived by his tests. He may, and often has, pronounced poison to be present when it was not; and he has overlooked it when it was present. What is produced as poison from a dead body, may not be poison at all. The varied results of chemical tests and processes may mislead, and often have misled the most experienced men; and there can be no doubt, that an absolute and blind trust in chemistry, as all-sufficient to settle a disputed case of death from poison in the affirmative or negative, would lead to the most serious consequences. The skilful poisoner, well acquainted with the selection of poisons, their doses and properties, and possessed of a knowledge of compounding them, would escape the penalty due to his crime, while the ignorant assassin who resorted to this species of murder would alone become amenable to the law. Abandon physiology and pathology as sources of evidence of poisoning, when chemistry fails, and no life will be safe. The path of secret murder will be clearly pointed out, to be trodden with impunity by those whose vicious propensities incite them to resort to these means of death. There is a refinement in crime, as there is progress in the arts and sciences; but it is only occasionally that we have to contemplate the perversion of medical knowledge and professional skill to the purposes of secret murder. Miss Abercromby, whose life was largely insured, died suddenly in 1830, having had symptoms of tetanus in a severe form before death. Chemistry failed to show any poison in her body, and the popular idea was then, as now—"no poison found, no death from poison." Her death was referred to cold and hysterical convulsions. No one who has considered the facts of this remarkable case, can doubt that this young



woman was poisoned by strychnia by her relative Wainwright. It was the first murder by strychnia perpetrated in this country; and, as I believe, completely overlooked by reason of this mistaken confidence in the infallibility of chemistry to reveal the presence of the agent under every conceivable case of death from poison. Twenty-five years have elapsed, and we have had another case under very similar circumstances. So long back as 1823, the French had practically settled the question which in this country led to the concealment of the murder of Miss Abercromby in 1830, and, but for a resolute and determined opinion, based on facts irrespective of chemistry, would have suppressed the murder of John Parsons Cook, in 1855, and ensured the impunity of the criminal. I allude to the case of Dr. Castaign, who was convicted and executed in 1823, for the murder of his friend Ballet by the administration of morphia. I shall, in another part of this paper, give a brief history of this case; but I may now remark, that the poisoning took place at a country inn—the intended victim was first dosed with tartar emetic—a medical man was called in—he was then dosed with morphia, from which he died. The scientific contest which took place in reference to Palmer's trial, occurred at the trial of Castaign. Six of the most eminent chemists in France made an analysis of the body, and found *no trace of poison*; and in this instance there had been no tampering with the stomach of the deceased. The defence was that the man had died of some obscure and unrecognisable disease, and that there could be no poisoning, when no poison was discovered in the body by men of great skill and experience in analysis. Only one physician, a man of eminence, could be found to sustain this doctrine; it was rejected by the court and jury, and the prisoner was justly convicted.

This false dogma was again brought forward at the trial of Palmer; and we may expect that in every case of skilful poisoning, in which, either by reason of the poison selected, by the mode of administration, or by subsequent tampering with the dead body and viscera, obstacles are thrown in the way of chemical research,—it will find its supporters. It is satisfactory to know, however, for the future security of life in this country, that, on the trial of Palmer, it received no support whatever

from any great names, whether in chemistry, physiology, or pathology, connected with the Medical Schools and Universities of London, Dublin, Edinburgh, Glasgow, Aberdeen, or St. Andrew's.

Although the result of this trial has shown that the popular view regarding the nature of the evidence required for conviction in a case of murder by poison is not correct, and that physiology and pathology may in some cases of themselves supply the deficiencies of chemical science, the question is still one deserving of grave consideration. The popular idea of infallibly detecting poison in the dead, has no doubt arisen from the occurrence of a large number of cases of poisoning by arsenic and other mineral substances, in the detection of which no difficulty has been commonly experienced, because, if the poison were lost from the stomach, it might be easily found in other and remote parts of the body, from which no art or skill could remove it, except by the complete destruction of the animal substance. We have yet, however, to acquire experience respecting the detection of strychnia. Chemical tests and processes have not undergone that ordeal with respect to strychnia which they have with respect to arsenic. This is the first case in which a person has been tried for murder in this country by the use of strychnia; and in this instance the perfect security and infallibility of the tests was not so much a question, as whether the poison could be detected under circumstances in which, up to the period of the murder of Cook, it never had been detected. When we find a chemist deposing on oath to the presence of this poison in a dead body in the proportion of a fifty-thousandth of a grain, and the conviction of the prisoner rests on the admission of this statement as evidence, we shall be better able to judge of the amount of confidence that will be placed by the jury, the profession, and the public, in chemical tests applied to such substances as strychnia, than we now are. This transcendental chemical evidence would in such a case lead from life to death, and would therefore require a little more sifting than it received at the late trial, where, as it was employed for the defence of the prisoner, it could only lead from death to life—that is, by its reception, to lead to the acquittal of the accused. It cannot be denied, however, that the conflict of scientific testimony

elicited on this and other points at the trial of this great criminal, has thrown disgrace upon the medical profession, and has created in the public mind a feeling of insecurity in relying upon scientific opinions under any circumstances. It will, I fear, take many years to remove this feeling; but, in the meantime, one great result has been gained. There is now no reason to fear that life will be insecure, because poison may not always be found in a dead body. A skilful assassin will not escape by the application of professional knowledge to the purposes of crime. At the same time, medical practitioners need not dread that a false charge will be raised against them because a patient happens to die suddenly under their treatment. William Palmer was not convicted upon loose coincidences of this kind, but, irrespective of all medical theories, he led to his own conviction by a series of acts which, in the mind of every unbiassed person, were perfectly inconsistent with his innocence.

On the 27th of November, 1855, Mr. Stephens, the stepfather of the deceased John Parsons Cook, was introduced to me by Mr. Warington, of Apothecaries' Hall. He was accompanied by Mr. Boycott, clerk to Mr. Gardner, solicitor, of Rugeley; and from Mr. Boycott I received a jar containing the viscera of deceased. It is proper to state that this jar had not at any time been in the custody of Mr. Stephens. I was informed by Mr. Stephens that the deceased, who was of the age of 28, and of average health, had died suddenly on the night of the 20th of November, 1855; that he had been slightly indisposed for about five or six days previously, the indisposition consisting chiefly in attacks of nausea and occasional sickness, but without purging; that on Monday night, the 19th of November, he had had a fit, from which he recovered; and that, on Tuesday, the 20th, about midnight, he was seized with convulsions, and went off suddenly. It was supposed that he had taken a pill, or some medicine which had been prescribed for him, on the preceding night; and I was further informed that an aged medical practitioner had been in attendance upon him shortly before his death. From circumstances which were not then stated to me, a suspicion had arisen in the mind of Mr. Stephens that his stepson had not died from natural causes; and, in order to set these suspicions at rest, he requested me



to undertake the analysis of the viscera, and to make a report of the results to him.

It subsequently transpired that, for about a fortnight before his death, and pending his illness, the deceased had been in company with a medical man,—a friend and associate, who was with him when he was first seized with sickness at Shrewsbury, when he was attacked with his fatal illness at Rugeley, and, lastly, who was present at the post-mortem examination of the body. The name of this person (William Palmer) was not mentioned, and it was not made known to me until after the analysis had been completed.

The deceased died on the 20th, and the examination of his body was made on the 26th of November. The jar containing the viscera was, as it has been already stated, delivered to me on the following morning. As the case appeared to involve the character, and possibly the life, of a medical practitioner, I requested that my colleague, Dr. Rees, should be associated with me in the analysis, so that, for the satisfaction of all parties, there might be a witness to the proceedings. This was assented to by Mr. Stephens.

Here it is necessary to pause, to show how the initiation of proceedings of this grave kind is sometimes a matter of accident, and how nearly one of the foulest murders of ancient or modern times altogether escaped detection. I was informed that an inquest on the body of Cook had been with difficulty procured by an appeal to the coroner: the examination of the body was made a matter of private arrangement on the part of the friends of the deceased, and Mr. Stephens was compelled either to allow the subject to pass over quietly, or personally to incur the risk and expense connected with the inspection and chemical analysis. The coroner was a friend of the suspected party, and if he had not placed obstacles in the way of a proper inquiry, he had certainly taken no steps to enforce it. He had no communication with me on the analysis; he had not seen that the post-mortem examination was placed in the hands of proper and responsible persons; and he had not taken care to prevent the presence of those who had a direct interest in throwing every obstacle in the way of a proper investigation. English coroners are not generally open to a charge of this kind: but on this occasion



Mr. Stephens, the stepfather of the deceased, was obliged either to forego a proper inquiry, or to pay out of his own means the costs connected with it. For the sake of public justice he adopted the latter course. He acted too with singular discretion in the matter. He took care that the viscera should be at no time in his custody, so that, in the event of poison being found, it might not be imputed to him that he had placed poison in the viscera owing to a vindictive feeling against the suspected person. The fact, however, that he had, at his own cost, procured an examination of the body and an analysis of the viscera, led to the improper suggestion, in the defence of the prisoner, that it indicated personal feeling. The fact was, however, and no one knew it better than the counsel who made this uncharitable suggestion, that had not Mr. Stephens taken this responsibility on himself, a triple murder, of a friend, of a wife, and probably of a brother, would have remained for ever concealed. The conduct of the coroner, in not taking upon himself the duty of procuring, for the sake of public justice, a proper examination and analysis, thus exposed a gentleman of unimpeached character to an undeserved and indecent attack, and created such difficulty in the subsequent analysis as practically to defeat it.

With the jar containing the viscera, a short note of the appearances found after death was placed in my hands. This note was in the form of a private communication from Dr. Harland, of Stafford, to Mr. Stephens. Dr. Harland was present at the post-mortem examination by the wish of Mr. Stephens, and furnished him with this private note at his particular request.<sup>1</sup> This note of the appearances threw no

<sup>1</sup> At the trial, this private memorandum was magnified by Sergeant Shee into a document most importantly affecting the interests of the prisoner, and of the knowledge of the contents of which they, the counsel for the defence, had been deprived up to that moment. The memorandum was delivered to me, by Mr. Stephens, with a request that I would not part with it unless it should be required for future reference or corroboration. It was in no sense a public document, but a private note. It was not signed by the other gentlemen present at the post-mortem examination, and merely had the initials of Dr. Harland. Although the learned sergeant might as reasonably have expected to receive copies of every private letter which had passed between Mr. Stephens and myself, he had not the candour to remove the

light on the cause of death. There was no trace of disease, or any morbid change in a vital organ, to explain the illness or nature of the convulsive attack under which the deceased was supposed to have died. The only appearances which particularly attracted attention, were the emptiness of the heart, and the general fluidity of the blood; but there was nothing to show the operation of any poison.

Such was the history of this case, as it was laid before Dr. Rees and myself, before we commenced the analysis, *i. e.*, within forty-eight hours of the time at which the viscera were delivered to me—the earliest period at which we could meet for this purpose. I subjoin from our notes, as an important preliminary statement, the following memorandum :

“ *Thursday, November 29th, 1855.*

“ In the jar was found the stomach, *cut open and turned inside out*, presenting over the greater part of its surface a deep reddish colour, especially towards the pyloric end. On examination by a lens, there was no ulceration nor whitish spots seen, nor was there any perforation. There was a feculent odour observed.”

The stomach was lying on the intestines; there were no contents; ligatures had been placed on the two ends of the stomach, probably with a view, in the first instance, of securing the contents, but for some reason, then inexplicable to us, this very proper design had been abandoned.<sup>1</sup> The jar contained only the stomach and intestines. We immediately

suspicion of unfair dealing which he had thus by his unfounded suggestion raised in the minds of the jury.

The reader will observe, that among the anomalies of this remarkable case, there was no regular report drawn up of the appearances presented at the post-mortem examination, and signed by all the persons present at it. This may account for some contradictions on minor points among the witnesses for the prosecution.

<sup>1</sup> Let the reader contrast this with the evidence given by Mr. Devonshire, who removed the stomach, and placed it in the jar. Having stated that he punctured the stomach, and some of the contents fell out on a chair—the question is put—

*Q.* Did you *tie* the stomach up *where it was punctured*, before you put it in the jar, with a *piece of string*.

*A.* Yes.

This evidence, it will be perceived, is perfectly irreconcilable with the state of

made application for the other viscera; and on the 1st of December we received, in a second jar, the liver, kidneys, and spleen, as well as a small bottle containing three drachms of blood taken from the vena cava.

The result of our examination is subjoined :

CHEMICAL EXAMINATION, MADE NINE DAYS AFTER DEATH AND  
FOUR DAYS AFTER INSPECTION.

*Stomach and Intestines.*—The stomach had been cut open, and it was found in the jar lying on the intestines turned inside out. The contents (if any) had entirely drained away from it. There was a brownish-coloured liquid adhering to some portions of the mucous surface or lining membrane. There was no appearance of ulceration, perforation, or other disease. The mucous membrane was generally reddened, and this redness was very strongly marked towards the pyloric or lesser end of the stomach. There was no appearance of effused blood under the mucous membrane; and on examining the whole surface of the stomach by a magnifying glass, nothing of a suspicious nature could be perceived. There was no mineral powder, nor any vegetable matter, deposited on or adhering to the surface. There was no odour of opium, prussic acid, or ardent spirits. The only odour perceptible was that of feculent matter, arising from the stomach having been placed in contact with the intestines in the jar.

The large and small intestines presented, both on the outside and on the inside, slight patches of redness in different parts. The small intestines contained a yellowish-coloured fluid, chiefly consisting of bile and mucus. The large intestines contained feculent matter, without any admixture of blood or any unusual appearance.

A large portion of the intestines was cut open, the contents washed out, and the intestines well drained in distilled water. The liquid thus obtained was found to be a mixture of bile, mucus, and feculent matter. After twenty-four hours, it deposited no mineral or vegetable sediment. This liquid was reserved for special examination. There was no blood contained in it or mixed with it.

*Liver, Kidneys, Spleen.*—The liver was healthy in structure. The kidneys and spleen were also free from disease. The left kidney was smaller than the right, and there was some slight phosphatic deposit in the tubuli uriniferi.

*Analysis.*—The washings of the stomach were first tested by the usual chemical tests and processes for arsenic, mercury, and other metallic poisons. They were found to contain a small quantity of antimony.

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the stomach when we removed it from the jar. There was no aperture tied up. There was no indication of an aperture having been made, for the stomach was completely cut open from one end to the other, and there was no string except the ligatures at the two ends. There is no reason to suppose that Mr. Devonshire has not stated what he actually saw and did; and therefore some person must have interfered with the stomach before it reached us. Otherwise, it could not possibly have been found as we found it.



The coats of the stomach, with the contents adhering to them, were examined for prussic acid, oxalic acid, opium, morphia, strychnia, veratria, nicotina, and conia, as well as for arsenic, mercury, antimony, and other mineral poisons. The result of this investigation was that they yielded only a slight trace of antimony.

The greater portion of the intestines, large and small, was submitted to analysis, and a small quantity of antimony was also found in them. The liquids of the intestines were examined for alcohol (distillation), and those poisons which might occasion sudden death, but there was no trace of any of these. Antimony was found in the fluids and coats of the intestines in small quantity. About one eighth part of the liver, the left kidney, and spleen, also yielded antimony. The quantity was less in the spleen than in the other organs.

The drainings of the jar in which the stomach and intestines had been placed were examined, and found to contain a larger proportion of antimony than the other parts. The drainings amounted to about two ounces, and consisted of blood and feculent matter mixed.

The contents of a bottle (3ijj) enclosed in the second jar, with the liver and kidneys, were found to consist chiefly of blood (putrid). They were examined for metallic poison, and yielded a small quantity of antimony.

#### CONCLUSIONS.

1. That *antimony* derived from some antimonial preparation taken during life, was present in the body of the deceased.

2. That antimony had been absorbed, carried into the blood, and deposited in the various structures in which it was found.

3. That the quantity discovered was small: it might be the residue of a large dose, or of various small doses, taken some hours, or even days before death.

4. That the quantity actually taken by the deceased, cannot be inferred from the small quantity found in the body of deceased, or from the appearances presented by the stomach and intestines.

5. That antimony under the form of tartar emetic may occasion nausea, violent vomiting, purging, and other symptoms of irritation of the stomach and bowels. In large doses it has caused death, and death has been preceded by convulsions.

6. That the viscera of the deceased, so far as they were examined by us, presented no appearances to account for death from natural causes.

7. That antimony as tartar emetic may be given as a safe and innocent medicine, or be used as a poison. We have no evidence before us to enable us to form a judgment as to the circumstances under which antimony was taken by deceased, and whether it was or was not the cause of death.

(Signed)

*Tuesday, December 4th.*

It will be perceived that the analysis left the cause of death quite undetermined. There was an absence of any accurate knowledge of the duration of the fatal illness under which the deceased sank, and of the nature, course, and progress of the symptoms preceding death. For anything that then ap-



peared to the contrary, the antimony found in the body might have been innocently given, and the deceased have died from natural causes, before the metal had been entirely eliminated.<sup>1</sup>

We were informed by Mr. Gardner, before we sent the report, that the medical practitioner suspected of having caused the death of the deceased, had recently purchased three active poisons, namely, strychnia, prussic acid, and Battley's sedative solution of opium. This information, however, was not allowed to affect or in any way alter or modify the conclusions at which we had arrived. It was enough for us that we had already performed our duty in searching, with the best means in our power, for the poisons indicated, with the result that there was no evidence satisfactory to our minds that any one of them was present. We did not, therefore, feel ourselves justified in assigning any cause of death from the facts before us. A private note to this effect, addressed to Mr. Gardner, was, as it was subsequently proved, opened by the postmaster of Rugeley, and the contents were communicated to William Palmer. It is proper to state, as much misapprehension has existed on this point, that the report of our analysis was not sent to Mr. Gardner at Rugeley, but to Mr. Stephens at his residence at Kensington. Had it been sent to Rugeley, it would no doubt have shared the fate of our letter: the seal would have been violated, and the contents communicated to the person since proved to have committed the murder.

It will be perceived from this statement, that as no poison was or could be specified when the viscera were delivered to us, our research was necessarily directed to the detection of a variety of substances which might occasion sudden death. We had only the *coats* of the stomach to deal with, and, in spite of

We thought that we had guarded ourselves from any imputation of rashness, by the cautious mode in which these conclusions were drawn up. We accused no one, but simply stated facts, and suggested possibilities which might be confirmed or refuted by evidence subsequently obtained. Let not a medical man, however, suppose that, by any amount of caution, he can protect himself from an imputation of this kind. When he has to deal with men who have no scruples as to the mode in which they conduct a defence, provided they can thereby gain a verdict, he must be prepared not only to hear his motives questioned, but his opinions denounced as rash and unjustifiable.

what some of the chemists retained for the defence assumed, we had not the *contents* before us. The drainings of the jar in nature and quantity did not correspond to the fluid which was said to have existed in the stomach, part of which was said to have escaped through an aperture made accidentally (!) in this organ during the inspection. Thus there were insuperable difficulties to the discovery of any alkaloidal poison, even supposing that all the material in the shape of a decoction of the coats had been consumed in the search for one poison, and that one *strychnia*.

Further, as there was clearly antimony in the body, and the quantity present might become a material question, we considered it to be our duty, after having made search for mineral poisons generally, to use the residue of the soft structures for the separation of antimony and for the detection of mercury. The search for the latter metal was made in consequence of the statement that calomel pills had been prescribed for and taken by deceased shortly before his death. The result was, that antimony was detected in all parts more or less, but there was no mercury. The quantity separated from the parts examined, was estimated at about half a grain.

After the report had been sent, a paper containing a statement by Mr. Jones, the medical gentleman who witnessed the death of the deceased, was placed in the hands of Dr. Rees, by Mr. Stephens, and from a consideration of this, assuming the statements to be true, it appeared to us, for the first time, highly probable that *strychnia* or *nux vomica*, in some form, might have been taken by the deceased, either by mistake or through design, and that this had really been the cause of the convulsions and death.

The inquest had been adjourned to the 14th of December, and on that day I attended at Rugeley, for the purpose of giving in evidence the results of our analysis, and, if possible, an opinion on the cause of death.<sup>1</sup>

After hearing read by the coroner, the deposition of Mr.

<sup>1</sup> Dr. Rees was summoned to attend the inquest, but owing to a delay of several hours in the delivery of the electric telegraph message, the cause of which has never been satisfactorily explained, Dr. Rees did not receive the information in sufficient time to attend, or his opinion at the inquest would have corroborated mine

Jones,<sup>1</sup> the medical friend who was present at the death of Cook ; the evidence of Mr. Bamford, who attended him and prescribed the calomel pills ; and of Elizabeth Mills, who alone saw the deceased in the fit on Monday night—respecting his condition on the five days preceding his death, the only conclusion that I felt could be drawn from the medical circumstances was that the deceased *had died in tetanic convulsions, and that these were caused by strychnia*. When this opinion was given, there had been no evidence that the prisoner had purchased strychnia. It was stated to the jury, in reference to the non-detection of this poison in the stomach of Cook, that, unlike arsenic, mercury, antimony, and mineral poisons generally, strychnia might destroy life in a very small dose, and that it was rapidly conveyed into the blood by absorption,<sup>2</sup> so that if a person survived an hour, none might be found in the body. When removed from the stomach by absorption, it had not been detected in other parts of the body. The symptoms, however, which it produced in the living body, were of so peculiar a kind, that these alone, as they were described to have occurred in the case of Mr. Cook, were sufficient to justify a medical opinion that death had been caused by strychnia. The fact that the stomach had been cut open, and that there were no contents sent for examination, except (as it was stated) such as were spread over the surface of some twenty

<sup>1</sup> Mr. Jones had been examined at the first inquest, and had given his evidence in my absence. Mr. Sergeant Shee, in addressing the jury for the defence, asserted that Jones had given his evidence in my hearing, and while I was present ; further, that I had suggested questions to bring out evidence that Cook had died from strychnia, and that until I had used the word "tetanus," Jones had not employed it. The whole of these statements are contrary to fact, as the learned counsel might have known, by the various documents before him. When reminded of his error by the learned Attorney-General, he said, "I did not mean to misrepresent anything!" The enthusiasm of a defence often carries a man far out of his depth. There was the same kind of misrepresentation (uncorrected however) respecting the evidence of the sale of strychnia, which he said had been given before I delivered my opinion. This also was untrue. Roberts, the witness to this fact, was examined some time after me. Misrepresentations of this kind give a false colouring to a defence.

<sup>2</sup> Experiments performed by Dr. Stevenson Macadam, since this trial, have proved that strychnia enters the blood, and is actually, in part at least, discharged from the body in the short period of *nine minutes*. ('Pharmaceutical Journal,' August, 1856, p. 124.)



feet of intestines, was already in evidence before them. This was a condition necessarily adverse to the detection of a poison like strychnia, even supposing that the question had simply been whether this poison alone was or was not present. Some of the chemists retained for the defence asserted, at the trial, that this mode of dealing with the stomach and its contents could make no practical difference, although it did not appear that any one of them had ever had a case in his own experience of searching for a variety of mineral and organic poisons, including strychnia, where the stomach had been thus cut, and the contents diffused, as alleged, over the whole intestinal surface. When such a case as this has been placed before them, they will be better able to judge of the difficulties, and to express less speculative opinions than those which they gave at this trial. They have endeavoured to teach the profession that carelessness in securing the contents of the stomach, or the stomach itself, in a case of suspected poisoning, is of no importance in reference to chemical results. It may, however, as any one will find by experience, render it impossible to detect an organic poison with certainty, or, if it be detected, it may render it impossible to say, when the answer to the question may be material, whether the poison found was contained in the stomach or in the fluids which had escaped from the bowels.<sup>1</sup> A quarter of a grain of strychnia diffused through two or three ounces of fluid in a dead stomach may admit of detection, supposing any of the poison to remain therein at the time of death; but when spread over or diffused through six

<sup>1</sup> Let the reader consider the rashness of such an opinion as this. Assuming that poison had been thus found loosely diffused over the intestines in the jar, this would not have proved that that poison had ever been in the stomach of deceased. It would be difficult, if not impossible, for a person intending to impute poisoning to another, to introduce a solution of strychnia into the stomach when removed and properly secured at the two ends; but what is there to prevent any maliciously disposed person from secretly pouring a solution of poison into a jar, where it may become diffused over the viscera, and so mixed up with the fluids, as to render it impossible to say whether it might or might not have been in the jar before the viscera were put in, or added to the viscera subsequently?

In a case, which occurred to me a few years ago, which was the subject of a trial for murder, the stomach of a child had been carelessly cut open, and the contents lost. The child had been destroyed by a decoction of hemlock. Not the slightest trace of any poison could be found.



or eight pounds of animal matter, including blood and fæces, it is an insult to common sense to assert that the analysis is not rendered infinitely more difficult and far less certain in its results.

At the time of the inquest, however, this question had not the magnitude which it afterwards assumed at the trial. It was considered sufficient to inform the coroner's jury, without entering into minute scientific details, that the circumstances connected with the death of Cook admitted of no other reasonable medical explanation than that it had been caused by one or more doses of strychnia, taken shortly before death. This conclusion was not, in my mind, in any degree weakened by the non-detection of strychnia in the body, because, with respect to this and some other poisons, my opinion was then, and is now, that we may more safely trust to pathology and physiology than to the crude speculations of chemistry.

It turned out, upon inquiry, that no antimonial preparation had been prescribed for the deceased by any of those who had attended upon him in a medical capacity since the first attack of sickness at Shrewsbury. How then came the antimony in the tissues of the body, in the contents of the intestines, and in the blood? The quantity found was small, but that was a point of little importance, since, except by the supposition of its having been long resident in the body, a conclusion opposed to its discovery in the fluid contents of the intestines and in the blood, it must have been taken by deceased shortly before his death,—a view supported by the fact that the nausea and sickness from which he had suffered during his illness at Shrewsbury and Rugeley could not be accounted for by any natural disease, while it was in accordance with the operation of small doses of tartar emetic, given to him occasionally by some person about him.<sup>1</sup> At the inquest but few questions

<sup>1</sup> As one instance among many of the ingenious modes in which the defence of Palmer was carried out, I may mention the following incident, communicated to me on good authority. Deceased was first taken violently sick, at the Raven Inn, at Shrewsbury, after drinking some brandy and water, which he thought had been "dosed" by the prisoner. Some time after the prisoner's committal, a paragraph appeared in a Shrewsbury paper, to the effect that, as an "extraordinary circumstance," a gentleman had been seized with sickness, like the late Mr. Cook, after drinking brandy and water at the Raven, and it was supposed that the liquors sold at that inn contained something noxious. The paragraph was a pure invention;

were put respecting the antimony. In answer to the coroner, I stated that the antimony found in the body might have been taken by deceased about eight hours before death, and that, from its general diffusion over the body, the conclusion of Dr. Rees and myself was that it had been taken some hours, and perhaps some days, before death. In answer to questions put by the jury, it was stated that the quantity of antimony found in the body would not have been too great to have been given as medicine, supposing that it had been administered by a regular practitioner. Vomiting causes a loss of a great portion of the dose swallowed. It would be scarcely tasted if given in wine or other ordinary liquid articles of food, when the dose was such as to cause nausea and speedy vomiting. The taste would depend on the quantity taken at a dose, and the mode of taking it. A person might unconsciously take a dose sufficient to cause nausea and vomiting. Antimony is not a substance which frequently destroys life, because it is seldom taken or given as a poison. A dose of eighteen or twenty grains might destroy life, but a person might die either from the exhaustion produced as a result of constant and severe vomiting or from the specially poisonous action of the antimonial preparation. Ten grains was the smallest dose which had hitherto been known to destroy life. Antimony should not be given in emetic doses in cases where there was a tendency to apoplexy; it might tend to cause effusion of blood on the brain. It would not be improper to administer it in cases where too much drink had been taken. Convulsions might precede death by antimony if the doses were large.<sup>1</sup>

and, on inquiry, was traced to a person strongly interested in the defence of Palmer. The object of causing such a paragraph to be circulated through the newspapers must be apparent.

<sup>1</sup> I subjoin a copy of my deposition at the inquest on Cook. After reading the report of our analysis, I said—"Therefore the result is, we find antimony in the body, but cannot account for the cause of death. The heart might have been emptied [as the result of] by spasm [either from disease] or poison. Antimony would not [be likely to] cause it [this appearance]. My opinion is that he [deceased] died from tetanus, and that this tetanus was caused by medicine administered or [and] taken shortly before death. I believe that the pills on the Monday night and the Tuesday night contained strychnia. I do not believe that the medicine administered [prescribed] by Dr. Bamford would have produced the effects I have heard [described] to-day. On the Monday night and the Tuesday night after the pills were taken, there was not the slightest indication [of the action]

Considering that in the case of the deceased there was nausea with occasional vomiting, soon after taking certain liquid articles of food, from the time that he was first seized with illness at Shrewsbury until his death, and that there was no natural disease to account for these symptoms,—the discovery of antimony in the viscera, as well as in the blood and contents of the intestines, appeared to us to be quite in accordance with a recent administration of this substance. There was no other explanation which would suit the facts; and although a suggestion was made subsequently, that the antimony might have been in the body for months and years, and even the whole duration of his life, there was not a shadow of reason to adopt this absurd hypothesis. The various articles of food taken by deceased acted alike in this respect. There had been no symptom of nausea or vomiting prior to the attack at Shrewsbury, and no evidence of deceased having taken antimony at any antecedent period. It would have been, therefore, in our opinion, most improper, when the discovery of antimony in the body was quite consistent with its recent administration, and with the symptoms proved to have existed in the case of the deceased, to have given a speculative opinion that the antimony *might* have been there for any conceivable period. Those who were inclined to adopt this hypothesis of remote deposition, had looked at only one half of the case. Assuming that the metal may remain deposited for several months in the liver, is it probable, after this long period, that it would be found in larger quantity in a free state in the blood and in the intestines? We, at the same time, gave no opinion respecting its lawful or unlawful administration; and

of morphia on the body. Further than this, we found no mercury in the liver or other parts of the body; and I do not think that mercury or calomel could have been taken on the Monday and Tuesday nights [as well as on the other nights] without our discovering [some] traces of it in the liver. The witness Mills has accurately described the symptoms produced by a small dose of strychnia such as would [might] be caused by pills [containing] strychnia given at half-past ten on Monday night; and the symptoms on the Tuesday night were those which would be produced by a larger dose of strychnia given in the pills taken on that night. There is an absence of any natural cause or disease to account for tetanus. The brain and spinal marrow were healthy. There was no insensibility before death; perfect consciousness, and merely that effect of spasm on the muscular system which a poisonous dose of strychnia would cause."



after hearing the evidence at the inquest, we felt that, medically speaking, death could not be assigned to the antimony found in the body. The circumstantial evidence subsequently given at the trial clearly proved that it had not been lawfully prescribed by any one in medical attendance on the deceased; and, while it tended to fix the administration of this substance on the prisoner, it fully confirmed the correctness of the opinions given at the inquest.

Much discussion has arisen respecting the object of the prisoner in administering antimony to the deceased. It is not often that two poisons are found in a dead body, or that two different substances are administered at the same time with criminal intention. Viewed by the light of facts connected with the death of the prisoner's wife, as they subsequently transpired at the inquest on Ann Palmer, they showed that one hand had been at work with the same instrument. Although this was a most important fact for public justice, the rules of law shut it out, and the accused had the benefit of the supposition that his wife had died a natural death, and that antimony had not been found in her body. The repeated doses of tartar emetic had, a year previously, succeeded in destroying the wife; and it may therefore be assumed that, in the case of Cook, the antimony was given in occasional doses, if not with the view of destroying his life, at least for the purpose of inducing an illness of longer or shorter duration, so that the death of the deceased should occasion no surprise by its suddenness. The illness thus produced by the antimony admitted, as occasion might arise, of the administration of some more powerful poison, which would speedily destroy life. It is remarkable that this has been the usual plan pursued by murderers who have acquired a knowledge of medicine. Some have supposed that, by the use of antimony in Cook's case, Palmer had speculated on causing the absorption and disappearance of the strychnia, or on defeating a chemical analysis; but it is highly probable that the antimony was used by him before he had even resolved upon the use of strychnia. It may be observed, that one effect of tartar emetic is, during the stage of nausea, to promote the absorption and rapid elimination of poisons; and by its emetic action to cause the expulsion by vomiting, of the residuary portion of unabsorbed



poison contained in the stomach. There was no evidence, however, that deceased had taken any antimonial preparation on Monday, the 19th November, the night on which the first and non-fatal dose of strychnia was administered to the deceased; and all the circumstances tend to show that the antimony was used simply with the view of causing some appearance of illness preceding death.

The coroner's jury did not undertake to solve the question as to the nature of the poison which caused death; they returned a general verdict to the effect, "That the deceased died by poison wilfully administered to him by William Palmer."

It was not until some weeks after the inquest, that I could procure a complete and connected history of the case of Cook, from the date of his first illness at Shrewsbury until his death. This, of course, appears in the evidence of different witnesses at the trial; but the account subjoined, divested of all extraneous details, will be found of great interest in a medico-legal point of view. It will enable the reader to compare the facts as they occurred in this case with the medical inferences drawn by the witnesses for the prosecution and defence.

#### MEDICAL HISTORY OF THE CASE OF JOHN PARSONS COOK, DECEASED.

Tuesday, November 13th.—The deceased, who was 28 years of age, had, generally speaking, enjoyed good health. He was at Shrewsbury races on the 13th, 14th, and 15th November, 1855. Mr. Jones, a medical man, in whose house at Lutterworth deceased had apartments, was with him (by invitation) there on the 13th. Jones dined with him that day. Deceased ate his dinner as usual; and when Jones left him, at 10 p.m., he was in his usual good state of health.

Wednesday, November 14th.—On this day deceased was still at Shrewsbury, in company with William Palmer and other sporting friends. Some brandy and water which he had on Wednesday evening caused violent vomiting. He saw a medical man there, who prescribed a pill and a black draught, and also a sedative mixture. He was seen to his bed-room that night by two friends, who state that they remained with him until 2 o'clock in the morning. He was frequently sick, but not purged. The next morning he was up, and able to attend to business.

Thursday, November, 15th.—Deceased, in company with William Palmer, arrived at Rugeley, from Shrewsbury, at 9:30 p.m. He told the servant, Mills, that he had been ill at Shrewsbury, and was poorly then. He went to bed at 10:30, without taking anything.

Friday, November 16th.—Deceased dined with William Palmer, and came home quite sober.

Saturday, November 17th.—Deceased did not get up to breakfast. He had coffee, and was sick immediately after he had swallowed it. He was frequently sick, and ascribed his illness to bile. Palmer sent him some broth during the day; deceased refused to take it, but Palmer insisted upon it. This was subsequently vomited. It appears that some aperient pills were given to him by Palmer in the morning. Mr. Bamford, the surgeon, first saw the deceased, by Palmer's request, at 3 p.m. on this day. He states that he found him suffering from violent vomiting; the stomach so irritable that it would not retain any liquid. He had been in that state for some hours. Pulse 70; skin moist; and the deceased was quite sensible. Deceased was in bed; he complained of no pain in the head, stomach, or bowels. Mr. Bamford prescribed an effervescing saline medicine.

About 7.30 on the evening of Saturday, 17th, Bamford was again requested by Palmer to see Cook. The sickness still continued. Prescribed for him, to be taken at night—*Morphiæ Acetatis*, gr.  $\frac{1}{2}$ ; *Calomelanos*, gr.  $\frac{1}{2}$ ; *Rhei Pulv.*, gr. iv; *Confect. Aromat.*, q. s., ft. pil. ij.

Sunday, November 18th.—Between 8 and 9 a.m., Palmer came to witness's house, and they went together to see deceased. He was then constantly sick. Deceased had had two or three hours' sleep in the night, but was sick on awaking. Witness remained ten minutes, and gave him some effervescing medicine in a neutralized state; it was ordered to be repeated every three hours. The liquid vomited in witness's presence was clear as water; no bile or blood, merely mucus and the liquids he had swallowed. [Mills, the servant, states that deceased vomited all Sunday morning. On Sunday evening he had a little barley-water, which remained on his stomach.]

Bamford states, that between 6 and 7 p.m. of Sunday, the 18th, Palmer came to his house, and asked witness to go with him to see deceased. They went: he was then sick, and had been sick all the day; the skin was moist; he had no pain, and was perfectly rational. Bamford further states that, on Saturday morning, deceased had taken, by Palmer's prescription, aperient medicine, and that this had acted both during Saturday and Sunday. The motions were natural; plenty of bile; there was no blood in them. Deceased complained of no pain on pressure; there was no tenderness over the region of the stomach.

It was on this day that Palmer wrote to Jones, the medical friend of deceased, who had parted with him in good health at Shrewsbury, on Tuesday, the 13th, the letter, of which the subjoined is a copy. It does not appear whether Bamford was made cognizant of this letter having been written.

“My dear sir,—Mr. Cook was taken ill at Shrewsbury, and obliged to call in a medical man; since then he has been confined to his bed here, with a very severe bilious attack, combined with *diarrhœa*; and I think it advisable for you to come and see him as soon as possible. Yours faithfully, (Signed), William Palmer, Rugeley, November 18th, 1855 (Sunday). ”

Monday, November 19th.—Bamford saw deceased between 8 and 9 in the morning; he then complained of sickness, but had not vomited. Prescribed a draught of Sulph. Magnesia, Infusion of Roses, and Tinct. Henbane, every three or four hours. Saw him again between 12 and 1. No nausea, no sickness; said the medicine had agreed with him; had had a basin of coffee, and broth. Bamford saw him again on Monday

evening between 6 and 7. He was then comfortable; had been up. The two morphia and calomel pills were delivered by Bamford to the servant at the inn. These pills were taken, or ordered to be taken, on the Saturday, Sunday, and Monday. There is no evidence that the pills, if taken on Saturday and Sunday, produced any inconvenience or disturbance, or unpleasant symptoms. On Sunday night, Bamford states that deceased wished to have his two pills (asked for them); hence it may be inferred that he had taken them, and derived benefit from them, on the two preceding nights. This day Palmer went to London, and returned to Rugeley in the evening.

Mills, the servant, states that on Monday, the 19th November, the sickness ceased, and deceased ate some breakfast and dinner, but continued very weak. William Palmer had been with deceased about 9 o'clock p.m. On Monday night, at about a quarter before 12 o'clock, witness was called to Mr. Cook, and saw him sitting up in bed, and beating the bed. He said, "Fetch Mr. Palmer." He had screamed twice. He thought the pills which he said he had taken about 10.30 had caused his illness. *His head was in motion, jerking backwards; his arms were straightened out, and his legs were set quite stiff; the eyes were staring; the head was drawn back; the mouth closed.* He could *talk*, and he said he should die. Palmer, who had then come over, said, "No, my lad, you won't." Palmer went for some medicine, and gave deceased two pills and some brown heavy-looking liquid—about a wine-glass nearly full. He was sick as soon as he had swallowed the medicine. They looked for the pills in the vessel, but could not find them. Mills remained with deceased until 3 o'clock on the morning of Tuesday, leaving Palmer with him. Deceased did not vomit again while she was there. He asked to have his hands rubbed; they were very stiff, cold, and damp from moisture. Seemed to recover. Deceased had complained of his throat, and thought he was suffering from cold. Palmer remained with him on Tuesday morning after she left. Went to the room about a quarter before 6, and Palmer was not there; he had then left. She did not see Palmer between 3 o'clock and 10 o'clock on Tuesday morning.

Tuesday, November 20th.—Mills saw deceased again at 7 and 8 o'clock; he would not take anything until he had seen Palmer. About 8 o'clock, deceased told witness he thought his illness was caused by the pills. Witness asked, about 10 a.m., whether he would have anything. He again said, "No; not until he had seen Mr. Palmer." At a little before 12 o'clock, deceased sent a message to Palmer, to know whether he might have a cup of coffee. Palmer said that he might, and that he would be with him directly. About this time Palmer appears to have come over. Witness made the coffee and left it with Palmer, who was then alone with Cook. Saw deceased two or three times during the afternoon. At 4 p.m. took him another cup of coffee. Left this also with Palmer, who, in half an hour, gave her a vessel, and said that Cook had vomited the coffee. Saw him several times afterwards that day. Gave him some arrow-root at 8 p.m. This was made in the kitchen. He did not vomit that. She was with him until nearly 10 p.m. He seemed lively and in better spirits. He talked of getting up the next morning; thought he should want the barber to shave him. He asked for some fresh toast and water.

[He appears to have shown no repugnance to drinking fluids, or difficulty in swallowing them.]

Mills had not gone to bed on Tuesday night when the second fit came on. She went for Mr. Palmer. He was over in three minutes (dressed).



Bamford did not see deceased on Tuesday morning. He states that Palmer called on him that morning; said he had been with Mr. Cook; that he was very quiet and comfortable, and *he did not wish him to be disturbed*. (It appears that nothing was said to Bamford about the severe fit on Monday night, or that deceased had ascribed this to Bamford's pills; or that he, Palmer, had sat up with deceased from 12 to about 6 o'clock.) Bamford, therefore, did not go near Cook on Tuesday until about 7 or 8 o'clock in the evening, when Palmer again called for him. In the mean time, Jones arrived. Jones, who had left deceased in good health at Shrewsbury on the 13th, arrived at Rugeley on the afternoon of the 20th, at 3 o'clock, and found deceased in bed. In reply to questions put by him, Cook said he was very comfortable, but very weak, and was not allowed to say much. He said he had been very ill. Mr. Palmer came in soon afterwards. Jones then examined Cook in the presence of Palmer; found his pulse soft and natural, and his *tongue clean*. Remarked to Palmer that it was hardly the tongue of a patient suffering from *bilious diarrhoea*, when Palmer said, "You should have seen his tongue before." [Bamford does not seem to have diagnosed bilious diarrhoea at any time; and Palmer had himself given active purgative medicines in pills on Saturday morning before Bamford was called to see deceased.] Jones saw him every half hour during the afternoon, sitting with him, and going down stairs occasionally. [It does not appear that Palmer informed Jones of the severe attack which deceased had had on Monday night, and which had led to his being called up and sitting with deceased for about five hours during the night.]

Mr. Bamford came with Mr. Palmer about 7 o'clock in the evening, and Mr. Bamford expressed an opinion that deceased was then going on *satisfactorily*.

Bamford says he put down the bedclothes and examined deceased particularly about the bowels and stomach, in the presence of Jones and Palmer. Deceased was then irritable, and appeared uncomfortable in his mind—distressed; turned his face from witness, who was on one side of the bed, while Jones and Palmer were on the other. The pulse was between 80 and 90, "full, irritable, firm, and trembling." Deceased said, I will have no more pills to night. He had asked for them on the previous night. It was agreed in consultation, however, that the pills should be given, and Palmer said to Bamford, I would rather you would make the pills again to-night. *He made them up in Palmer's presence, wrote a direction, at Palmer's request ("Night pills"), and delivered them to Palmer, who took them away with him.* Bamford did not again see the deceased alive.

Jones states that at the consultation it was suggested by Palmer that Cook should not know what the pills contained, as he strongly objected to them on the previous night, because they had made him ill.

Palmer came with the pills about a quarter past 11 o'clock. He produced two pills, and gave them to deceased in Jones's presence, calling Jones's attention to Bamford's handwriting. Almost immediately after swallowing the pills, deceased vomited. It was a momentary act. The pills could not be seen in the chamber-vessel. Deceased then lay down, and was quiet. He dreaded an attack like that of the previous night, which he described as a kind of fit. Witness went to bed in the same room, at a few minutes before 12. At that time deceased appeared as comfortable as usual. At about ten minutes past 12, Jones was suddenly roused by deceased, who had jumped up in bed, saying, I am going to be ill, ring the bell for Mr. Palmer. He asked witness to rub his neck. Soon after Palmer came (*i. e.*, in



about two or three minutes). He brought with him two pills, which he gave to the deceased. He said they contained *ammonia*. Immediately after taking these pills, deceased screamed loudly, and threw himself back on the bed, in convulsions. He asked to be raised up, saying, "I shall be suffocated." They endeavoured to raise him, but he was so stiffened out with spasms, that it was impossible to do so. When he found we could not raise him, deceased said, "Turn me over." Jones then says, "I turned him on his right side, listened to the action of his heart, which gradually ceased, and in a few minutes he died. I have never seen symptoms (convulsions) so strong before. They were symptoms of *tetanus*; every muscle in the body was stiffened. When deceased asked to have his neck rubbed, which was in about three minutes after I was first roused, I found that his head and neck were affected with spasms. His head was thrown back, his hands were clenched, and his arms were in a state of rigidity. His jaw was fixed and closed. His body was stretched out, and resting on his head and heels (opisthotonos)."

[It thus appears from Jones's statement, that at 12 o'clock, and until about ten minutes past 12, the deceased Cook was in a comfortable state. In three minutes, *i. e.* at 12:13, the spasms in the head and neck were strongly developed. After this, Palmer came, and deceased asked him for the remedy he had taken the preceding night. The remedy, *i. e.* the ammonia pills, were taken in about ten minutes, making the time 12:20. Deceased lived six minutes after swallowing the two pills brought by Palmer, making the whole duration of the case from sixteen to twenty minutes, death having taken place at 12:26, or thereabouts.]

*Inspection.*—This was made six days after death, in the presence of Palmer and others. The viscera were universally in a sound state. The membranes of the brain were a little congested (*dura mater*). The heart empty; the blood generally fluid. No appearances in any part to account for death. The body remained in rigid spasm after death, and was so found in the extremities on exhumation, more than two months after death.

*Analysis.*—The only discovery made was, that absorbed *antimony* had been deposited in, and was contained in the tissues of the stomach, intestines, liver, kidneys, spleen, and blood.

The cause of death, on the one hand, is assigned to strychnia given in the pills taken at a quarter past 11 on Tuesday night; on the other, to some natural disease—idiopathic tetanus or hysteria.

The deceased had two attacks, one on Monday night, and the second, or fatal one, on Tuesday. They were precisely similar in their character, but that of Tuesday was more severe. The attacks came on about *an hour* after some pills had been taken by deceased on *each* night. He had perfectly recovered on the Tuesday morning, and remained in a very comfortable state during the whole of that day, and in fact until, without any assignable cause, the symptoms suddenly appeared, and killed him *under tetanus and opisthotonos of the most formidable kind*, in *sixteen*, or at the furthest, *twenty minutes* from the commencement of the attack.

Can it be reconciled with idiopathic tetanus, or with any known disease of the nervous system, that it should thus appear, without any assignable natural cause, on two successive nights, that there should be a complete intermission of the tetanus for nearly twenty-four hours, and that it should then again burst forth and prove fatal in the short period mentioned?

The fate of the prisoner at the subsequent trial depended on the answer to this question. But there was another question closely connected with the above, on which great difference of opinion was likely to arise; namely, whether any description of symptoms or medical circumstances could justify a medical witness in assigning death to poison when he was unable, by analysis, to reproduce from the dead body, in a visible and tangible form, some portion of the poison alleged to have caused death.

As the supposed natural disease, if any, would probably have affected the spinal marrow—and the upper part only of this organ had been examined—it was considered desirable for the ends of justice, that a more minute examination should be made. The body was exhumed about two months after death (January 25th, 1856), and it was then ascertained that the spinal marrow and the vertebral canal were quite healthy,—that there was no change of appearance in these parts, which could be assigned to disease during life. There was nothing, in short, in the condition of this organ which would account for the sudden attack of tetanus and death. Two medical gentlemen attended the inspection on the part of the prisoner; and they had full liberty to take away any, or the whole of the body, for the purpose of verifying the accuracy of the analysis already made, as to the presence or absence of antimony and strychnia. Only a small part of the spinal marrow and its membranes was, however, taken by them, with a view to a pathological examination, and not to a chemical analysis. This is a significant fact, and it tends to throw an important light on the chemical portion of the defence, and on the *bona fides* with which the scientific question of the presence or absence of strychnia in the body of Cook, was subsequently treated.

To my apprehension, the symptoms under which Cook died, as described at the inquest, were quite irreconcilable with any known form of disease. This judgment was not formed by taking fractional parts of the case, or adopting the practice pursued by some of the medical witnesses for the defence at the trial, namely, of making comparisons of individual symptoms in different cases. The case was viewed as a whole;

and as such, it stood out clearly and distinctly as a case of poisoning by strychnia.

The symptoms indicative of tetanus as a result of strychnia in a fatal dose are—trembling of the muscles, shaking of the body, stiffness of the back of the neck, general uneasiness, and a feeling of suffocation. The body and extremities are suddenly stiffened by muscular spasm; the hands are clenched; the toes are incurvated, the soles of the feet becoming hollow; the body then assumes a bow-like form; it rests on the head and heels, and the back is arched; the muscles of the abdomen are hard. During the paroxysm there is severe pain, leading to shrieks and cries, from the almost universal cramp; the face is dusky or livid, the lips are livid; the mind is clear; there is consciousness. Remissions of these symptoms occur at short intervals; but, after a succession of the fits, and sooner or later in proportion to their intensity and duration, the person dies either from asphyxia, or as a result of exhaustion of nervous power, by reason of their frequency and severity.

I here transcribe from my notes, written long before the trial, a summary of the reasons on which my opinion of the cause of death in Cook's case was based:—

*1. Reasons for the Opinion given at the Inquest, that Cook died from the effects of Strychnia.*

The symptoms were undoubtedly such as a dose of this poison would cause. They came on very suddenly about an hour after some pills had been swallowed. These pills should have contained morphia and calomel, and have produced a sedative effect on the body—calming any excitement and producing sleep. On the Saturday and the Sunday night the pills appear to have had a good effect; at least, no injurious symptoms were produced by them—no convulsions, no spasms. If Bamford, therefore, prepared the pills from the same bottles, and according to the same prescription, on the four nights which preceded death, he could not have made a mistake, and put strychnia for morphia, apart from other facts stated by him at the inquest, showing that he could not have mistaken his bottle of strychnia for that containing morphia.

On the Monday and Tuesday nights only do these pills act differently. Instead of the calmative effects of morphia, there are the violent symptoms of strychnia. The deceased himself ascribed his symptoms on Monday night to the pills which he had taken, and refused to take any more.

While there is no indication of such effects on the living body as the morphia pills ought to have produced, and had apparently produced on the two previous nights, there is no trace of mercury to be found in the liver after death. Had the four doses of pills containing calomel been swallowed on the four successive nights, it is in the highest degree probable that some trace of mercury would have been found in



the liver. I state this, on a remarkable case which occurred to me some years ago, in which some months after death I detected mercury in the liver of a person who had taken only a few grains of blue pill shortly before he died. It would not be proper, however, to rely greatly on this point. It might fairly be open to doubt and objection. I would rather base an opinion on the symptoms which actually followed in the case of Cook, being so entirely different from those which ought to have followed, supposing the pills to have contained morphia and calomel, and not strychnia.

Before leaving this part of the case it should be stated, that, subsequently to the inquest, Dr. Rees and I analysed the contents of a pill-box found in prisoner's room, and detected therein a preparation of mercury (calomel?), and a substance resembling morphia in its chemical reactions. The substance was broken up, and did not correspond in weight to Bamford's pills.

*Time of Occurrence of Symptoms.*—The symptoms came on, on each night, *i. e.* (Monday and Tuesday), about an hour after the pills had been taken. This is quite in accordance with the operation of strychnia. When the strychnia is dissolved, the symptoms may come on earlier. The time at which they appear after the poison has been taken, may vary according to the state of constitution, and many other circumstances. When the poison has been absorbed in sufficient quantity, they begin to show themselves, and the rapidity of absorption probably varies in different persons and in different states of the body. The pills taken by deceased on the Monday night could not have contained sufficient strychnia to destroy life: but the symptoms were severe, and lasted some time. From one quarter to one half of a grain might account for the effects on Monday night; and three quarters of a grain to a grain might account for the symptoms and death on Tuesday night.

The effects produced on Monday night would enable a person acquainted with the properties of strychnia to adjust the fatal dose with tolerable accuracy on the Tuesday night.

## *2. Reasons for the Opinion that Cook did not die from any form of Tetanus as a result of disease.*

There is no doubt that the actual cause of death was tetanus in a very severe form. The question is, therefore, what was the cause of the tetanus?

Tetanus, which implies a general spasm, or cramp of all the muscles of the body that are usually under the power of the will, may arise from,—1, poison; 2, wounds (lacerations, severe bruises) (traumatic); 3, exposure to cold and wet (idiopathic).

As to *poisons*—arsenic, antimony, and other irritant poisons, may occasionally produce tetanic spasms of the muscles, but then there are always other symptoms which precede or follow, of a totally different kind.

Strychnia is the only poison (with the exception of brucia) which produces tetanus in a pure and unmixed form, and this was tetanus in its pure and unmixed condition.

In Cook's case there was no wound or personal injury; hence it was not a case of what is called traumatic tetanus. Idiopathic tetanus is comparatively a rare disease, and not very often fatal.

There was no exposure of the deceased to wet or cold. He remained from Wednesday night until the following Monday without any symptom indicative of tetanus, until an hour after he had taken the pills on Monday night. Bamford saw



nothing to indicate tetanus on the Saturday and Sunday. There is therefore (apart from the pills) no cause to account for an attack of tetanus in the case of Cook; and being a case unexampled in the severity of the symptoms, and its rapidly fatal termination, on the assumption of its being idiopathic tetanus, there ought to be some apparent cause for its origin. There was no proportionate excitement to account for it, and the morphia and calomel prescribed by Bamford would have tended to allay such, had it existed.

## DIFFERENCES.

*Idiopathic Tetanus from Exposure to Cold and Wet.*

1. Symptoms have no connection with any liquid or solid swallowed.

2. Symptoms commence slowly, and progress slowly; difficulty of swallowing; stiffness of jaws, of neck; after some time the body, the legs, lastly the arms; hands not commonly affected.

3. Opisthotonos, or body bent back in the form of a bow, resting on head and heels; does not come on until after many hours or days from the attack.

4. Paroxysms, or fits of spasm, may be severe, and the person may die from exhaustion. Patient commonly recovers after some days or weeks.

5. In idiopathic or traumatic tetanus there is no *intermission* in the symptoms, merely a remission of the paroxysms. The patient is always under the influence of the morbid cause, which remains until he dies or recovers.

*Tetanus from Strychnia.*

1. Some solid or liquid taken within about two hours or less of commencement of symptoms.

2. Symptoms commence suddenly with great violence. Nearly all the voluntary muscles of the body are simultaneously affected. Arms and hands spasmodically clenched at the same time as body and legs. Jaw not primarily affected, not always fixed.

[N.B. Cook was able to swallow and speak within ten minutes of his death. No case of idiopathic tetanus, so far as I can find, has presented this condition, the jaw being generally the first part to become fixed.]

3. Opisthotonos, a very early symptom, in a few minutes commonly.

4. When symptoms are once clearly established, they progress to death or recovery. They occupy only minutes. In from ten minutes to two hours after commencement, the person dies or recovers, according to the severity of the paroxysms, and strength of his constitution.

5. In tetanus from strychnia, if the dose should not be sufficient to prove fatal, the effects pass off; patient recovers; there is a complete intermission in the symptoms.

[N.B. This is a remarkable feature in Cook's case; since, on the Tuesday, he had quite recovered from the attack on the Monday night.]

The differences here assigned, show that in the case of Cook the tetanus was produced by something administered, and not by any ordinary cause.

It may be objected to the medical opinion, that Cook died from an attack of *hysteria* simulating tetanus.

#### DIFFERENCES.

##### *Hysteria.*

1. Connected with a peculiar constitution, chiefly seen in females.
2. Patient subject to previous attacks or fits.

The spasms may be tetanic in hysteria, but there are more commonly convulsive motions of the limbs, alternating with stiffness or rigidity, generally a loss of consciousness, and other symptoms of hysteria.

3. Paroxysm not fatal; patient speedily recovers.

No instance, so far as I know, is recorded, in which a young and healthy man in the prime of life, has been at once (suddenly) seized with a fit of hysteria, presenting only the features of tetanus from poison in its most severe form, and like it, proving fatal in twenty minutes from the first attack or commencement of symptoms.

##### *Tetanus of Strychnia.*

1. Not connected with any peculiarity of constitution.

2. Cook had not been subject to such attacks at any period; never had had any kind of fit before this. A healthy active young man in the prime of life, given to out-door pursuits.

If occasionally subject to excitement, the effects (if any) would supervene immediately, and not be postponed for several days. There was no cause of hysterical excitement on Monday and Tuesday night.

3. He speedily dies in a paroxysm.

##### *Post-mortem Appearances.*

There is nothing of a peculiar character in these appearances, whether the tetanus depend on disease or on strychnia.

In Cook's case, the principal appearances were,—1, fluidity of the blood; 2, emptiness of the heart; 3, some congestion of the dura mater, or outer membrane of the brain.

All that can be said is, that these appearances are consistent with death from strychnia; and further, they fail to show in any part of the body any *natural cause* for this violent and sudden death. The spinal marrow was subsequently found healthy.

There is not much probability that a case of tetanus from disease could be mistaken for tetanus from strychnia-poisoning. The risk is almost entirely the other way. Dr. Watson justly observes, our eyes should be open to the possibility of a case of poisoning by some of the preparations of strychnia, being palmed upon us for a case of natural

disease.<sup>1</sup> This form of poisoning, he remarks, may be easily mistaken for tetanus. The rapid progress of the symptoms, and speedy death, have always been regarded as strong indications of poisoning. There is a case of traumatic tetanus, as it is called, quoted by one author from another, on the authority of Prof. Robinson, of Edinburgh, who died about the end of the last century, which it is said proved fatal in a quarter of an hour. The anecdote is this:—The professor was once at table, when a negro servant lacerated his thumb by the fracture of a china dish. He was seized with convulsions *almost instantly*, and died with tetanic symptoms in a *quarter of an hour*. It is highly probable, as Dr. Watson observes, that assuming the statement to be true, the man died rather of fright than of traumatic tetanus. The average period of the access of tetanus from a wound is from the fourth to the fourteenth day. In the above case, the man was a negro, and negroes have been observed to be particularly liable to tetanus from slight causes. There was a lacerated wound also: hence, admitting the authenticity of the case, it could not be compared with that of Cook, for there was no injury to his body which could account for the occurrence of tetanus. An attempt was made to introduce this case, but no stress was laid upon it. I subjoin a statement of the most rapidly fatal cases of tetanus, as it was prepared for evidence at the trial, also a statement of some cases in which tetanus followed and proved fatal after ulcers. It was quite clear, from the evidence of Dr. Savage and others at the trial of Palmer, that Cook had no ulcers to which tetanus could be reasonably referred, and if he had had ulcers at the time of death, the fact that he was seized with tetanus and died in twenty minutes would have been quite inconsistent with all experience on the fatality of tetanus from such a cause. The supposed ulcers could not have been a cause of tetanus on the Monday night; and after this, there was complete recovery for twenty hours before the second and fatal attack. Nevertheless much was said about ulcers, and there was obviously an intention on the part of the defence to present this to the jury as a satisfactory explanation of the tetanus under which deceased sank.

In Case 102 in a table compiled by Mr. Curling, tetanus

<sup>1</sup> Practice of Physic, i, 558.



ended fatally in twelve hours. In Dr. Laurie's table of 41 fatal cases, in the Glasgow Infirmary, the shortest period in which the disease destroyed life was twelve hours after amputation of the thigh. In a boy, whose case is detailed in Mr. Curling's work, the disease proved fatal in sixteen hours. The above cases were traumatic.<sup>1</sup>

In Dr. Laurie's table of 50 traumatic cases, in the Glasgow Infirmary, and in his table of 171 published cases of traumatic tetanus, one in the former, and two in the latter, occurred after *ulcers*. Mr. Hutchinson, 'Medical Times, vol. xxix.' gives the case of a man (Case 31) having an ulcer on the leg, of unknown duration, in which death occurred after a single day, with symptoms of tetanus. Fournier Pescay very accurately describes, in the *Art. Tetanus*, in the Dictionnaire de Médecine (1821), the case of a soldier, who wounded the little finger with an axe: he was seized with tetanus in three hours, and died in fourteen hours after the accident.

In March, 1856, a case of tetanus, arising from ulcers, was brought into the London Hospital. This proved fatal in from eight to ten hours. The details were given in evidence at the trial of Palmer by Mr. Ross. A man, æt. 37, having chronic indurated ulcers, two at the back of the right elbow, near the ulnar nerve, and one on the left, was brought into the hospital at half past seven in the evening. He was then breathing quickly, the jaws were closed; he was unable to swallow; the muscles of the abdomen and back were somewhat tense. In ten minutes he had a second paroxysm (having previously had one in the receiving room), with opisthotonos, which lasted about one minute; he was then quiet for a few minutes; he had then another fit, in which he died, having been in the hospital only half an hour. On cross examination, it was elicited that the symptoms in the jaws had come on about *nine hours before his admission*, and that there had been convulsive paroxysms all the afternoon. Mr. Ross had no doubt that the man died from tetanus, and that the cause of the tetanus was referable to the indurated chronic ulcers described. Thus, then, this was simply proved to be a fatal case of tetanus from ulcers. The stomach of deceased was examined for strychnia, but of course none was found. Was there, indeed, anything about the case that could have led to the

<sup>1</sup> See Appendix to this paper.



supposition that the symptoms were caused by strychnia? Was the treatment, on admission, such as is immediately resorted to in cases of strychnia poisoning? In the first place there was no evidence that the man had swallowed anything or had been able to swallow anything since the time at which he had had his dinner, *i. e.* about eight or nine hours before his admission. There was no reason to suspect an intention to commit suicide; or that his wife, who came with him to the hospital, had intended to poison him. Again, the convulsive symptoms commenced and progressed very slowly, so that eight hours had elapsed before they had acquired great severity, and the man lived, from the date of their commencement, the long period of nine hours, or thereabouts. There was a visible and satisfactory cause for this tetanus in the old chronic indurated ulcers under which the deceased had been suffering for twelve years. The symptoms began, as in most cases of traumatic tetanus, with stiffness, followed by spasm and fixedness of the jaws; the abdomen, back, and extremities being only long afterwards affected; there was inability to swallow; and although the arms and legs were convulsively affected in the paroxysms preceding death, it does not appear that there was any clenching of the hands, or incurvation of the feet. Apart from the absence of all moral evidence, there was not a single feature in the commencement, progress, and duration of the symptoms in this case, by which it could be confounded with a case of strychnia poisoning. Although more rapidly fatal than traumatic tetanus is usually observed to be, it would have been a case of uncommon duration for one of strychnia-poisoning. In tetanus from disease, the case is reckoned by hours or days, in tetanus from strychnia, by minutes. There was not one point of resemblance between it and that of Cook.<sup>1</sup>

<sup>1</sup> The subjoined paragraph, in reference to this case, appeared some time before the trial of Palmer, in the Birmingham and Staffordshire papers, and was afterwards copied into some London papers. This may furnish a key to its being produced in evidence. It would of course find circulation among the class of men from whom jurors are selected.

"An extraordinary death from tetanus has taken place at the London Hospital. Edward White, aged thirty-seven, was brought in with symptoms as of poisoning by strychnine; his wife said he had been ill some days, and he died a few hours after admission. An inquest was held, at which Dr. Letheby stated that he made an analysis of the deceased's stomach and its contents. He searched for strychnine in

I willingly admit that it would have saved Dr. Rees and myself from much obloquy and vituperation, had we assigned tetanus, in Cook's case, not to strychnia, but to the idiopathic disease from cold taken on the race-course at Shrewsbury, to angina pectoris, or to a sudden attack of "epilepsy with tetanic complications," depending upon some unrecognisable disease of the spinal marrow,—ignoring at the same time the presence of antimony in the body, and referring the nausea and sickness from which deceased had suffered to chronic irritation of the stomach, brought on by intemperance. Nothing would have been more easy than to have given an opinion, such as that which was given in the case of Miss Abercromby, that cold and hysteria would account for tetanus and sudden death. In short, from the circumstances so favorable to the accused under which this inquiry was conducted, there would have been no difficulty whatever in withdrawing from public notice Palmer's crime.<sup>1</sup> The reward for acting with decision and firmness on this occasion has not been such as I think will induce professional men to follow my example in any similar case

consequence of the symptoms, but found no poison at all. Since so much had been said about strychnine, witness had made numerous experiments with it, and could now detect the hundredth part of a grain if it had been present. The cause of death was tetanus. It was an extraordinary case, and he knew of no other on record. A verdict was found "the deceased died from tetanus, but how caused there was not sufficient evidence to show."

<sup>1</sup> The emptiness of the heart was, in my absence, dwelt upon by the coroner, in his final address to the jury, as a condition inconsistent with the opinion which I had given of the cause of death: because *a case* had been described by me, in my work on 'Medical Jurisprudence,' in which the heart and lungs were found gorged with blood. I had already, however, answered the questions put to me on this point, and had quoted to the jury another case of poisoning by strychnia, in which the heart was found empty. The coroner is reported to have impressed upon the jury, that Mr. Bamford, who was present at the death of the deceased [an error], thought that he had died of apoplexy, and "even after the brain had been cut open," he was of the same opinion! With a jury of more pliant character, this would have had the effect desired by Palmer, in his letter addressed to the coroner shortly before.—"I hope the verdict, to-morrow, will be, that he died of natural causes, and thus end it." If the coroner believed that the published views of a witness were inconsistent with his evidence given at the inquest, it was his duty to point out this inconsistency to the witness when present, and not reserve his comments for a time when the witness was absent. It would then be out of the power of the witness to explain the differences, or to satisfy the jury that such inconsistency did not exist.

hereafter. There is, however, only one plain duty to be performed on such occasions, however painful it may be. Whatever may be the consequences to the medical jurist, although he may stand alone, and have the attacks of the public and medical press directed against him, the safety of society, and the demands of public justice, require that an opinion formed deliberately after a careful consideration of all the medical circumstances, should be firmly and decidedly expressed. After all, it must be remembered, the medical opinion refers to the cause of death, rather than to the criminal. The medical facts may admit of only one reasonable interpretation, namely, that the person died of poison; it must be the conduct of another, his motives and his actions, irreconcilable with innocence, which will ultimately fix the perpetration of the crime upon him. The medical opinion of the cause of death in Palmer's case, had no relation to the prisoner's conduct—in his assigning a false cause for the illness; in his endeavouring to procure a hurried interment of the body; in his interfering at the post-mortem examination, and cutting into the jar containing the viscera; in his attempting to bribe a post-boy to overturn the carriage and break the jar; in opening, or causing to be opened, letters having relation to the results of the analysis; in writing to his friend the coroner, expressing his desire that the inquest might be brought to a close with a verdict of death from natural causes, &c. I might add to these circumstances the purchase of strychnia, without any assignable or reasonable motive for procuring this formidable poison on two different occasions within twenty-four hours of Cook's death. It was not the medical opinion respecting the cause of death, or, as I have elsewhere remarked, any medical theory regarding the operation of strychnia, which led to the conviction of the prisoner at the trial, so much as these portions of his conduct, for which the subtle and overstrained arguments of his counsel could afford no reasonable or satisfactory explanation. They were, in fact, left without an answer.

The view propounded by Mr. Sergeant Shee, however, was that the medical opinion of the cause of death given at the inquest, was based, not upon the symptoms, but on the knowledge that strychnia had been purchased by the prisoner. In the kind of defence set up on the occasion of this trial, at



which every circumstance, medical and moral, adduced by the prosecution, tended to establish the guilt of the prisoner, it would have been vain to expect a charitable, or even a reasonable view of the motives for medical opinions. It happened, however, unfortunately for this part of the case, that the symptoms suffered by Cook on the Monday night, were equally referred to strychnia, although there was then no proof of the purchase of the poison by the prisoner until midday on the Tuesday, the day on which the deceased died. Besides, as I have elsewhere stated, the fact of the purchase on the Tuesday was communicated to Dr. Rees and myself, before we had even sent our report; and that report, as it is well known, did not assign death to strychnia or to any other cause.

There was no evidence of the possession of strychnia by the prisoner on Monday night, until the day before the trial, *i. e.* five months after the opinion had been given at the inquest, that the symptoms on that night were distinctly due to strychnia. On this point, therefore, the defence utterly failed.<sup>1</sup>

I need not remark that the opinion given at this inquest, regarding the cause of death, was fully confirmed at the subsequent trial, by the evidence of Sir Benjamin Brodie, Dr. Todd, Mr. Solly, Mr. Curling, Dr. Daniel, Dr. Christison, Dr. Rees, and others. With the views so clearly expressed by these witnesses on the differences between the symptoms of tetanus as a disease, and those caused by strychnia, and their concurrence in the opinion, that while the symptoms manifested by Cook on the

<sup>1</sup> This part of the defence, involved a striking inconsistency :—the medical opinion that the symptoms were caused by strychnia on the Tuesday night was alleged to have depended on a knowledge that the poison had been purchased on that day; but the medical opinion, that the symptoms on the Monday night had arisen from precisely the same cause, had been formed and given without any knowledge of the possession or purchase of the poison, and maintained for five months in the absence of any proof to that effect.

Some have thought that the procuring of three grains of strychnia, at Rugely, on Monday night, was an act which would not have been done by a guilty man. The poison, however, was *not purchased*, but procured as a gift from the house of a person where drugs and chemicals were not sold. As this was not a retail shop, no inquiry was made there, and none knew, or could possibly know, that poison had been given, except the giver and the receiver. Had it been procured at a retail chemist's in London, or elsewhere, the purchase would have been soon traced by the police. Therefore this mode of procuring the poison on Monday was well adapted to conceal the transaction.



Monday and Tuesday nights were quite consistent with the effects of strychnia, they were not in accordance with those of any known form of disease, I consider it quite unnecessary to discuss this question further. The chief witnesses for the defence were compelled to admit that the symptoms were such as strychnia might produce, and the reason probably why Sergeant Shee, did not call—Mr. Lawrence, surgeon of St. Bartholomew's Hospital, who attended in court, for the defence, for several days; Dr. Williams, Professor of Materia Medica in the Royal College of Surgeons in Ireland, and surgeon for eighteen years to the city of Dublin Hospital; Dr. Nicholas Parker, of the London Hospital (both of whom, he promised, in his address to the jury, to place in the witness-box), Dr. Marshall Hall, Dr. Copland, Dr. Carpenter, and others—was that they would in all probability have confirmed substantially the evidence of the witnesses for the prosecution, and have given an opinion that the symptoms of Cook were those of strychnia, and not such as they had ever witnessed in tetanus from disease. With regard to Drs. Copland and Watson, they were easily accessible as witnesses if they could have testified in favour of the prisoner; but the learned sergeant preferred relying upon selected extracts from their published works! The other witnesses who were examined for the defence, did not agree among themselves; one assigned one cause of death, and one another;—thus the jury were left to select some convulsive disease, modified epilepsy, hysteria, arachnitis, epilepsy with tetanic complications, and angina pectoris; one professed that he was unable to assign any cause for the symptoms, either natural disease or poison; but he thought that “some peculiarity of the spinal cord, unrecognisable except the examination be made immediately after death, might produce symptoms like these;” a very convenient hypothesis for covering any kind of death from poison affecting the nervous system, when from circumstances the poison cannot be discovered in the body. It simply implies that a *recent* examination of the spinal cord sets all questions at rest respecting the pathological seat of convulsive diseases; and that hydrophobia, hysteria, tetanus, and epilepsy, are at once indicated by the inspection of this organ, while the body is recent!

One of the strongly contested questions at the trial—involving many of the points just considered—was this :

WERE THE SYMPTOMS AND APPEARANCES IN THE CASE OF COOK CAUSED BY STRYCHNIA, OR MIGHT THEY HAVE ARISEN FROM SOME NATURAL DISEASE ?

On this question, as it has been already remarked, the evidence of all the witnesses for the prosecution was clear, distinct, and conclusive ; and the three cases of death from strychnia—namely, those of Agnes French, Mrs. Sergison Smyth, and Mrs. Dove—supplied all that was wanted to make out a complete history of poisoning by strychnia in the case of John Parsons Cook. Certain objections to this conclusion were taken by the witnesses for the defence, on the ground that some of the symptoms in Cook's case were inconsistent with poisoning by strychnia ; and it may now be desirable to consider how far these objections are well grounded. The dissentient views expressed at the trial were chiefly derived from what had been witnessed of the effects of strychnia on animals. One witness thought that the very *sudden accession* of the symptoms was greater in the case of Mr. Cook than in strychnia-poisoning. According to my experiments, and those of others, the symptoms in animals generally come on suddenly, after some slight uneasiness. But observations on the human subject are more to the purpose.

In the case of Agnes French, detailed at the trial, the symptoms came on suddenly, and the patient was found in violent convulsions. (See No. 4 in the table of fatal cases at p. 347.) In the case of Mrs. Smyth (No. 10 in the table, p. 350) they also came on suddenly, and the ringing of a bell, which was considered to be a difficulty in Cook's case (although this took place on the Monday night, and not on the Sunday), was also an act performed by Mrs. Smyth, after she had taken a fatal dose of strychnia. The *sitting up* and *beating of the bed* (the *malleatio*<sup>1</sup> of Sergeant Shee), and the screaming, observed

<sup>1</sup> *Malleatio*. This learned word was applied to that part of the evidence of Elizabeth Mills, in which she states, she saw Cook "sitting up in bed, and beating the bed." Hooper, in his dictionary, tells us, that it is a form of chorea, in which the person has a convulsive action of one or both hands, which strike the *knee* like

in Cook's case, as well as the power of having his neck rubbed without causing spasms, are quite consistent with poisoning by strychnia. The first two acts are those to which a man complaining of a feeling of suffocation from any cause would be likely to resort, and may, like the screaming, precede the access of spasms. They were, in fact, felt by the patient himself to be the forerunner of an attack of spasms, and described by him as such. As to the screaming and tolerance of the *rubbing* of the body, Mrs. Smyth's case (No. 10, p. 350) affords an example of this. A case published by Dr. Lawrie and Dr. Cowan shows that, in poisoning by strychnia, this rubbing may not only be borne, but give relief.<sup>1</sup> It is absurd to describe Mrs. Smyth's as an exceptional case, merely because great sensitiveness of the skin is generally manifested in the poisoning of *animals* by strychnia. The record of cases in the human subject does not bear out the view that human beings are similarly affected, and the statement that animals are *always* so sensitive that they will not bear rubbing without the production of tetanic spasm is not in accordance with well-observed facts. Drs. Lawrie and Cowan justly remark that it is "the sudden first impression on touch, and not the *continuous pressure*, which causes the shock and the renewal of the spasm,—a circumstance which appears to have been entirely overlooked by the witnesses at Palmer's trial." \* \* \* "We have uniformly found," they observe, "that rubbing any part of the surface in dogs does not cause spasm; and that, so far as we could judge, rubbing or scratching their necks was agreeable to them." As the Attorney-General remarked, the dogs and rabbits which were the subject of experiment could not ask to have their necks rubbed or manifest the "*malleatio*;" and, although much reliance was placed on their acute sensitiveness to slight touches, under the operation of strychnia, not

a hammer. A more probable interpretation is, that Cook was in a distressed state from a feeling of oppression, and a sense of suffocation, and that he was simply doing what patients frequently do under the same circumstances, throwing his arms about on the bed. Why such a learned term should be applied to so simple and natural an act as this, it is difficult to understand, but for the fact, that it was thereby raised into the dignity of a special symptom, by which Cook's case was to be distinguished from other cases of strychnia-poisoning. The ringing of a bell might as justly have been transformed into a symptom, under the name of "*tintinnaculatio*."

<sup>1</sup> 'Glasgow Medical Journal,' part xiv, July, 1856.



one witness appears to have tried the effect of rubbing or stretching the muscles, or it would probably have been found that it could have been borne, as in the cases of the animals thus treated by Drs. Lawrie and Cowan.

A question, however, here suggests itself. Does it follow that the acute sensitiveness observed in animals should also be observed in man? May not the poison affect man and animals differently in this respect? Trismus is a common and early symptom in animals. It is not usually observed in human beings until after the paroxysms of convulsions have set in, and there may be equally a difference with respect to sensibility. Devergie observes that all poisons which affect sensibility, whether local or general, have not the same influence on man and animals, because it is impossible to establish the least comparison between the sensibility of the dog and that of a human being.<sup>1</sup> According to Pereira, the exaltation of sensibility in man is rather the effect of a succession of large doses used medicinally, and acting perhaps with accumulative force, than of one fatal dose. It is to be remarked that the observations of the witnesses, so far as they referred to the human subject, were confined to cases in which strychnia had been used medicinally. They had not seen one case of acute poisoning by strychnia like that of Mrs. Smyth or Mr. Cook.

The next important objection is based on the act of *vomiting*. Mr. Nunneley is asked by Mr. Sergeant Shee—"Is there any medical reason that occurs to you why the patient (poisoned by strychnia) should *not* vomit? *A.* I apprehend, where there is so much spasm, there is an inability to vomit: in cases related where attempts to vomit have been made, they could not succeed. I have *a case*, which is related in the tenth volume of the 'Journal de Pharmacie,' in which attempts were made to give emetics without success."

Dr. Letheby is asked by the same counsel—"Is the *vomiting* of the pills *before death* inconsistent with what you have known or observed in strychnia poisoning? *A.* It is not consistent with anything I have observed."

The act of vomiting by Cook here referred to must have been on the Monday night—in the course of the slighter attack from which he completely recovered. On the Tuesday night,

<sup>1</sup> Toxicologie. Tom. iii, p. 47.

at the time the pills containing the strychnia were administered, Jones stated there was a momentary act of vomiting; but this was an hour before any symptoms had commenced—in fact, just as the poison had reached the stomach. There was no vomiting of the pills before death, or while Cook was under the violent symptoms which killed him, and the question and answer were based on a complete mistake.

Assuming, however, that vomiting had really occurred, Sergeant Shee was well aware that Cook had had frequent attacks of sickness during his illness, and that antimony had been found in his body, even in his stomach. He should have therefore asked his witnesses these further questions: “Q. Supposing the animals on which you experimented had been previously well dosed with antimony, would it not be likely that this antimony would give rise to great irritability of the stomach? A. Yes.—Q. Then you would not feel surprised at an effort in the stomach to reject pills, swallowed most unwillingly, whether they contained strychnia or any other substance? A. No.”

But the answers of the witnesses as they stand, are perfectly inconsistent with the facts observed in strychnia-poisoning. In the case reported by Drs. Lawrie and Cowan, already referred to, notwithstanding the existence of spasms arising from a dose of three grains of strychnia, the patient swallowed three or four doses of an emetic, which was passed to the back of the mouth, and *free vomiting was induced*. This was two hours and a quarter after the ingestion of the poison, and *three quarters of an hour after the first spasm!*

In case No. 2 in the table (p. 346), in which a very large dose of strychnia had been taken, slight vomiting was produced by tartar emetic. In Case No. 11 (p. 350), in which one grain of pure strychnia had been taken in a pill, from which the deceased died in one hour and a half, there was violent sickness; and in a case reported by Dr. Beck,<sup>1</sup> in which half a grain of strychnia caused spasms with opisthotonos, there was occasional vomiting.

Facts of this kind may not be consistent with what the witnesses have themselves observed, but they prove incontestably that the act of vomiting is not inconsistent with poisoning by

<sup>1</sup> ‘American Journal of Med. Science,’ Oct., 1851, p. 535.

strychnia, even supposing that the vomiting had occurred at the time in which it was incorrectly represented to have occurred in the case of Cook. One other assumed difference is also strongly dwelt upon by the witnesses for the defence, namely, *the length of time which elapsed before symptoms came on* (about an hour or an hour and a quarter), although evidence had been given at the trial that a protraction of symptoms had been distinctly observed in a case of poisoning by strychnia for the period of one hour after the poison had been taken.

Mr. Nunneley and Dr. Letheby both assign this long interval as one of the reasons why they would not ascribe the death of Cook to strychnia. Their experience, they admitted was chiefly derived from experiments on animals. Mr. Nunneley had experimented on sixty animals, and the time of occurrence of symptoms from the ingestion of the poison was from *two to thirty minutes*, more generally about *five or six*. Dr. Letheby stated that he had seen "some dozens" of cases of the administration of strychnia to animals, and the average time when the symptoms began was a quarter of an hour. He had seen them begin in five minutes, and the longest interval was *three quarters of an hour*. In answer to a question, he said, "I have never witnessed such a long interval between the administration of the poison and the coming on of the symptoms as in this case."

The case No. 3 in the table (p. 347), of which a report was published in the 'Lancet' for August 31st, 1850, p. 259, was given by me in evidence at the trial. It is that of a girl who had taken one grain and a half of strychnia. *No symptoms appeared for rather more than an hour after taking the poison.* In 1848 Dr. Anderson published a case of poisoning by strychnia, in which a man took, by mistake for muriate of morphia, three and a half grains of strychnia. No symptoms appeared for *two hours and a half*.<sup>1</sup> In the case of Drs. Lawrie and Cowan, already referred to, which occurred on the 11th June, 1853, an adult took three grains of pure strychnia, perfectly dissolved in a mixture of spirit, sulphuric acid, and water. Like Mr. Cook, he went to sleep, and about *an hour and a half* afterwards he suddenly awoke in a spasm, uttering loud

<sup>1</sup> 'Edinburgh Monthly Journal,' February, 1848, p. 566.



cries, which alarmed the household.<sup>1</sup> The witnesses for the defence were no doubt correct in saying that in their experiments on animals, they had never met with a longer interval than forty-five minutes; but the above facts in reference to the human subject, one of which was actually in evidence before the Court, shows the extreme danger of a witness attempting to solve a question of this kind by little or no experience on the human subject, and by a limited experience on animals. Even so far as animals are concerned, the statement is extraordinary. In one experiment by Dr. Christison, no symptoms appeared, in a rabbit, after a fatal dose for *an hour and three quarters*; and in an experiment by Dr. Rees and myself, in which one grain of strychnia was given to a rabbit, there were no symptoms for *an hour and five minutes*. Some facts have been communicated to me in which five or six hours have elapsed before symptoms appeared in animals. In fact, it generally depends on the dose, on the rate of absorption, on the mode in which the poison is given, and various other circumstances. It must be, however, perfectly clear that with experience accessible from other sources, there was not the slightest justification for the statement that the interval which had elapsed before the symptoms showed themselves in Cook, was so great as to be any real objection to their having arisen from strychnia. In questions having this general bearing on scientific truth, witnesses are not expected to rest on their own experience; and certainly no case was ever tried in our Courts, in which so great a freedom of reference to all facts connected with poisoning by strychnia, was conceded to witnesses. Mr. Nunneley, indeed, did not hesitate to refer to a case, in the '*Journal de Pharmacie*,' in support of his view, that spasm would prevent vomiting in strychnia poisoning.

So far with the objections from *symptoms*. The witnesses when asked respecting *appearances* in the dead body, stated that the condition of the heart in Cook, was not consistent with death from strychnia. Dr. Harland deposed that "*the heart was contracted, and contained no blood.*" In the private memorandum sent to me, the only notice of the state of this organ was—"the heart was of natural size, and in every part

<sup>1</sup> See the case, '*Glasgow Medical Journal*,' part xiv, July, 1856.

healthy." The blood is stated to have been fluid in all parts of the body.

It was admitted by the witnesses on both sides, that in their experiments on animals, the heart was generally found full of blood, especially on the right side; but the question arises, whether a point of this nature can be determined better by the results of experiments on animals, than by observations on man. On reference to the table of fatal cases of strychnia (p. 346), the variable nature of the appearances in the body, will be at once apparent. Age, sex, the dose, state of health, and peculiarities of system, will account for the differences which appear, and if such differences exist among human beings, *à fortiori*, they may exist between human beings and animals. A sufficient number of facts, however, have been accumulated to show that "animal experience" would lead to very fallacious conclusions. In case No. 2 (p. 346), in the table, fatal in one hour and a half, the *heart was found flabby and empty*,—the *blood every where fluid*. In case No. 4 in the table (p. 347), that of Agnes French, given in evidence at the trial, in which the patient died in about an hour, the *cavities of the heart were quite empty*, the muscular fibre of the heart was stiff. In case No. 10 (p. 350), that of Mrs. Smyth, also given in evidence before the Court, *the heart was contracted and perfectly empty*. In case No. 13 (p. 351), in which the patient died in about an hour after taking one grain and a half of strychnia, the heart was found, by Dr. Lonsdale, to be healthy, *empty and unnaturally atonic*. In No. 14 (p. 351), death from a large dose; the heart is described as flabby, and containing *only an ounce of blood in the left ventricle*. In No. 15 (p. 352), we are told by Dr. Geoghegan, an accurate observer,—a case in which five grains of strychnia had proved fatal in twenty-five minutes—that *the heart was firmly contracted*, and its cavities contained a *very small quantity* of dark fluid blood. Thus, out of *ten inspections* in *fifteen* fatal cases of poisoning by strychnia, the heart was either found empty, sometimes contracted, and sometimes flabby, or there was not that fulness of the cavities which, from observations on animals, the witnesses had pronounced to be a necessary attendant on poisoning by strychnia!

To these facts other cases might be added; for instance, that of Mrs. Dove, although the emptiness here was explained

by one of the witnesses as arising from the draining of the organ by reason of the head having been first examined. This does not appear to afford a satisfactory explanation. In cases Nos. 10 and 14, in which the heart was entirely or nearly empty, the head was not examined; and in cases, 5, 8, 12, in which the cavities of the heart contained much blood, the head had been examined. In an American case, of which I have a private note, that of Mr. G. W. Greene, tried at the Chicago Circuit Court, December, 1854, for the murder of his wife by poisoning her with strychnia, it was stated in evidence by Dr. Freer, who examined the body, that the heart of the deceased *was healthy, but empty*, as well as the large vessels near it. In the case of Azenath Smith, tried in Canada, in 1851, for the murder of her husband by administering strychnia in pills, Dr. Dupson, who gave evidence on the occasion, said that he found the heart of the deceased, *empty in all its cavities, and healthy*.<sup>1</sup>

In a recent case of poisoning by nux vomica, which is, in fact, poisoning by strychnia, reported by Mr. Davies, the same condition of the heart was observed. The quantity of nux vomica taken was half an ounce, and the patient died in two hours after the commencement of the symptoms. The body was inspected seventy hours after death, and among other appearances, he states that "to the touch the heart felt quite hard; its structure appeared quite healthy; its smallness may be accounted for *by the whole of its cavities being empty*. The *auricles and ventricles did not collectively contain a drachm of blood*."<sup>2</sup>

These facts are sufficiently numerous to show that the empty condition of the heart found in Cook, is perfectly consistent with death from strychnia-poisoning, as it has occurred in human beings, however it may appear to militate against the results of observations made on animals. My own experiments, on a few rabbits, are in accordance with those of the witnesses for the defence, who have had the advantage of a larger experience in this direction; one having put to death by strychnia upwards of sixty animals, including dogs, cats, rats, mice, guineapigs, rabbits, frogs, and toads; the other having

<sup>1</sup> 'London Medical Gazette,' September 19, 1851, p. 517.

<sup>2</sup> See 'Medical Times and Gazette,' February 9, 1856, p. 149.



destroyed the indefinite number of "some dozens"—"the right side of the heart was always full of blood." At the same time, there appears to be some difference even among animals. M. Startin, who has recently published Dr. Booth's case (No. 1 in the table, p. 346), states that it was not possible in that case to procure permission to examine the chest. From experiments on dogs, instituted at the time by himself, he states that both the *full* and *empty condition* of the *heart* may be found, and that this was accounted for in the animals by death taking place either during an inspiration or during an expiration.<sup>1</sup>

This question is obviously of great importance in regard to diagnosis in future cases. Is this emptiness of the heart to be or not to be taken as one of the appearances likely to be met with in poisoning by strychnia? Is it a sign of some other fatal disease; and if so, of what? It cannot be referred to idiopathic asphyxia. The cases already collected of strychnia-poisoning, to which may now be added that of John Parsons Cook, show, whatever may be the results of observations on animals,<sup>2</sup> that emptiness of the heart, with sometimes flaccidity and sometimes tonic contraction, is a state which has been frequently observed in death from strychnia, and thus it may be met with in future cases. I am indebted to Mr. Poland for some information respecting the condition of the heart in death from idiopathic or traumatic tetanus.<sup>3</sup>

Of the occasional congestions found in the membranes of the brain and spinal marrow, in the substance of those organs and of the lungs, it is unnecessary to speak. There was nothing in respect to these parts in the case of Cook, which was at all opposed to the supposition of death by strychnia.

<sup>1</sup> 'Medical Times and Gazette,' July 12, 1856, p. 36.

<sup>2</sup> If there are differences in the *symptoms*, there may be differences in the *appearances*, produced by strychnia on man and animals. According to my experiments, spasmodic closure of the jaw sets in among the early convulsive symptoms in animals. This is certainly not the case in the human subject. According to observation, it appears late. Should we be justified in inferring from this effect on animals, that fixed closure of the jaw is one of the first symptoms of strychnia-poisoning in man,—and because in Cook's case, the jaw was not closed, and he was able to swallow "antidotal" pills within ten minutes of death,—that his symptoms were not referable to strychnia?

<sup>3</sup> The reader will find this information in an Appendix to this paper.

The only conclusions which it appears to me we can draw from this inquiry are—

1. That the symptoms and appearances observed in the case of John Parsons Cook are not only consistent with death by strychnia, but such as from experience in other cases in the *human subject* we may commonly expect to find as a result of the action of this poison.

2. That the symptoms and appearances, taken as a whole, are not reconcilable with death from any other cause.

3. That the objections urged to this cause of death by the witnesses for the defence have no real foundation, and are opposed to observations hitherto made in cases of death from strychnia.

By a comparative examination of the table, it will be seen that the case of Cook (No. 16, p. 352) is in close accordance with the general order and progress of the symptoms, as well as with the appearances met with after death in the fifteen fatal cases which are reported. If a medical witness insists upon selecting isolated symptoms or appearances for comparison, he will find an ample field for dispute in comparing known cases of poisoning by strychnia with each other. There are some in the table which differ more from each other than Cook's case differs from them. If he ignores facts, already recorded by others with no other view than that of contributing to the general stock of scientific knowledge, if he persists in confining his opinion to his own limited experience of cases in the human subject, or if, in ignorance of reported cases in human beings, confines his inferences to results obtained from experiments on animals, he may be so far accurate in his evidence, but he will at the same time seriously mislead a Court of law. A jury placing reliance upon such partial evidence as this, might be led to return a verdict quite contrary to the truth. The fact is, a scientific witness is called not merely to inform the court on what he has himself seen, done, and known; but, in addition, to give the general results of scientific experience from all authentic records. It is only by this that the truth can be really ascertained.

The solicitors for the prosecution in this case, therefore,

acted wisely in placing before the jury, in all their details, and irrespective of any medical theories or conclusions derived from experiments, three clear and unambiguous cases of poisoning by strychnia. A suggestion was made on the part of the defence, to the effect—that in the facts in which these cases strongly resembled that of Cook, they were of an exceptional kind (e. g., Mrs. Smyth's case) ; but the information thus laid before the jury enabled them to throw aside much of the medical sophistry with which the evidence had been overlaid, and to take a common-sense view of the facts. A strong complaint has been made on the conflicting opinions given regarding the cause of death ; but such conflicting opinions must always be given if medical facts are either not known, only partially known, or wholly ignored. It is impossible, under such circumstances, that there can be agreement or consistency in the evidence. There is no resource but to test the accuracy of an opinion, not by the individual experience of the witnesses, but by the general experience of the medical profession.

The next important question which arose out of the evidence given at this trial was the following :

CAN A PERSON DIE FROM POISON AND NO POISON BE FOUND BY CHEMICAL ANALYSIS IN THE BODY.

I here put this question generally. At a very early period in the progress of this case, it was found that if the defence failed in assigning the symptoms of Cook to some latent disease, the only point on which it could rest was this:—If it be alleged that a person has died of poison, let it be produced in a visible and tangible form ; if it cannot be produced, then, supposing proper skill to have been employed, the only inference to be drawn is, that no poison was taken, and that death was caused by disease. This is bringing the question of death from poison to a very simple issue indeed. It is casting aside physiology and pathology, and requiring our law-authorities to place entire and exclusive confidence in the crucible and test-tube of the chemist. But has Organic chemistry, with all its modern advances, yet reached a point that no death can occur from poison, speaking generally, except the poison be still



found either in the stomach, the tissues, the blood, the excretions, or in all of these parts at one and the same time? Is the viper-poison easily revealed by tests? Can the poison of rabies, producing one of the most formidable convulsive affections known, namely hydrophobia, be detected in the tissues? Is there any chemical process by which the poison of the ordeal bean of Africa, or even of the common laburnum, the seeds of the *Ricinus communis*, the poisonous fungi, darnel, and the sausage-poison of Germany, the poison of the *cœnanthe crocata*, can be separated and demonstrated to exist after death in the blood and tissues? If not, then the allegation that no person can die from poison except the poison be found in the body is a mockery and a delusion, admirably adapted to cover a multitude of deaths from poison, which, but for this dogma, might be revealed by pathology and physiology. It is all the more dangerous, because the history of crime shows us that the arts of the murderer, especially of the scientific or professional murderer, are daily becoming more refined. I might add largely to the list of poisons which either by their nature, by their tremendous power in very small doses, or by the mode in which they are introduced into the system, might infallibly produce death without leaving a physical or chemical trace of their presence in the body. I forbear to do this. Such an enumeration would undoubtedly serve my purpose of refuting that which I believe to be a gross and dangerous error on the part of some of the chemists who gave evidence for the defence at the trial of Palmer; but it would be at the cost of making public, means of death and modes of perpetrating crime which it would be dangerous to promulgate. The fallacious doctrine here broached for the temporary purpose of saving the life of a wretched criminal was, however, such as to receive, at least for a time, a large amount of popular support. There was an astonishing plausibility about it, especially in the form in which it was almost daily circulated in newspaper paragraphs emanating from the solicitor for the defence or his agents. It only required bold assertion, and the chemical differences hitherto admitted by chemists to exist between organic and inorganic poisons at once vanished. Alarm was also spread and allowed to pervade the public mind by the allegation, that unless poisons were invariably detected and separated, in cases of

alleged poisoning, any innocent person might be convicted of murder by poison when the death was really due to some latent disease. It is fortunate that the jury in Palmer's case have, by their verdict, given the death-blow to this novel and dangerous doctrine, and have shown that twelve men may be as safely directed to a just decision by the views of pathologists and physiologists as by the assumptions of chemists. This is as it should be. Chemistry may detect a poison ; but it fails, without the aid of physiology and pathology, to show whether it was or was not the cause of death ; and, in some instances, it cannot enable us to determine whether the poison was introduced into the body during life or after death. Even with regard to the poison in question in this particular case,—*strychnia*,—this substance is now so extensively employed as a medicine, that the discovery of traces of it in the stomach, blood, and tissues (assuming that the processes used are satisfactory) would not justify an allegation of death from poisoning by it. *The symptoms must be made known.* The “tetanic complications” which it ordinarily produces in the body when taken in poisonous doses must be clearly established, and a judgment must be based on these symptoms. We are not, therefore, to suppose, as the public have been erroneously led to imagine, that toxicology and chemistry are convertible terms, that the finding of poison in a body is a proof of death from it, and the not finding the poison is a proof of death from some natural cause.

It is, I think, satisfactory to science that no physician or chemist of any authority as a toxicologist, could be found to support this novel doctrine. I have reason to know, that some men highly distinguished for their chemical and medico-legal knowledge, in the three kingdoms, had been urgently pressed, on the part of the defence, to come forward and maintain that the discovery of poison in the body was a *sine quâ non* in every case of alleged poisoning. It is highly creditable to them that they positively refused, on this ground, to appear in the case. Only two gentlemen at all known to the public could be found to support this view ; namely, Mr. Herapath, of Bristol, and Dr. Letheby, of the London Hospital, both of whom were, by a singular coincidence, engaged for the defence in the case of another great criminal, Tawell, who

was convicted and executed for poisoning Sarah Hart, by prussic acid, at the Aylesbury Lent Assizes, in 1845.<sup>1</sup>

Dr. Christison, a writer of deservedly high authority, and a witness for the crown in Palmer's case, says, in reference to the question which we are here considering, that the doctrine that "no charge of poisoning can be established without the discovery of poison in the body or in the evacuations, is a great error. Under this doctrine few criminals would be brought to justice were they to resort to a variety of vegetable poisons, which, in certain seasons, are within the reach of every one."<sup>2</sup> Dr. Geoghegan, Professor of Medical Jurisprudence in the Royal College of Surgeons, Ireland, remarks, that "the information derivable from a chemical examination of the contents of the stomach, although, of course, a most important element in medico-legal investigations connected with poisoning, does not carry with it that exclusive weight which is attached to it by those of the profession who are uninformed in forensic medicine; nay, further, although a positive decision may be occasionally arrived at from other single sources of evidence, the *chemical* examination alone cannot decide the cause of death."<sup>3</sup>

<sup>1</sup> In this case, my friend the late Mr. Cooper, who conducted the chemical analysis, informed me, that he procured a *grain* of anhydrous prussic acid by distillation from the stomach of the deceased, and he gave evidence to this effect at the trial. This was quite sufficient to account for death. The presence of the poison could not be disputed; but it was here contended, of course by the sanction of the chemists, that this quantity of prussic acid had been spontaneously generated by a chemical reaction in some apple-pips, which the deceased was supposed to have swallowed with the pulp of apple, found in the stomach. The defence was essentially of a scientific and chemical nature. It utterly failed, because it was opposed to common sense and common experience. A medical witness may, however, safely rely upon the fact, that when there is a sufficient stimulus for the display of chemical ingenuity in "shaking" the evidence of witnesses engaged in a public prosecution, it will always be forthcoming. If the poison is not found in the body, then it will be urged that there is a total want of proof of poisoning; if it be found, the witness will have to meet the objections, that there was not enough in the body to account for death, or failing this, that the tests and chemical processes employed, were not satisfactory; in fact, not such as "higher adepts" in science would resort to, or lastly, if this position should be untenable, then that the poison was the product of some spontaneous chemical changes within the body, and that it was not administered to the deceased.

<sup>2</sup> 'Treatise on Poisons,' 4th edition, p. 71.

<sup>3</sup> Case of Poisoning by Monkshood, 'Dublin Medical Journal.'



In accordance with these views, the great principle of jurisprudence in a criminal charge, now confirmed by the verdict in Palmer's case, is not that a poison should be invariably found in the dead body, but that there should be satisfactory evidence of death from poison; and this may be had when chemical tests, even in the hands of "experts," fail to reveal its presence. To affirm that innocent persons may be placed in danger from the adoption of such a principle, is absurd; it is equal to affirming, that on a question of poisoning, chemistry alone is to be relied on; and that persons accused of this crime are to be convicted or acquitted according to whether a chemist believes, by the production of certain colours, that he has extracted the fifty-thousandth of a grain of some alkaloid from the thirty-second part of a liver, or that he is prepared to swear to the presence of the substance from the form of a crystal seen under a polarizing microscope, and calculated to weigh the ten-thousandth part of a grain. The danger, in my opinion, is quite the other way. By placing a blind reliance upon such extreme results, the accuracy of which cannot be determined, or their value judged, by courts or juries, there is great risk that a person may be condemned on erroneous evidence. At any rate, his only chance of safety would be in the corrective proofs derivable from physiology and pathology; that is to say, in the nature of the symptoms preceding death, and in the appearances presented by the dead body. On the other hand, as this extreme nicety of detection is not admitted to be a property of organic poisons by men of experience and repute as toxicologists, who, in their scepticism, can have no object but the public good, it follows that many criminals would escape, if such a doctrine were once admitted by our Courts of law.

It has been supposed that Palmer's case presented a novelty in this respect, and it was rather industriously asserted, in certain publications, emanating from the defence, that no case could be pointed out in which there had been a conviction for murder by poison and the poison not found in the body. The case of Dr. Castaign, who was tried and executed in Paris, in 1823, for the murder, by poison, of his friend and companion, Augustus Ballet, presents many points of resemblance to the case of Palmer, even in the kind of defence set up. Castaign

had been a pupil under Orfila, and was charged with having made use of his knowledge of poisons to take away life. The prisoner was intimate with two brothers, who were young men of fortune. One of them, Hippolyte Ballet, who had been for some time an invalid, and was attended in his illness by his friend Castaign, died rather suddenly, in October, 1822. No suspicion then arose that he had died from other than natural causes. He had made his will in favour of Castaign, bequeathing nothing to his brother. Castaign afterwards surrendered the will to the surviving brother, Augustus Ballet, for a large sum of money. About seven months after this, the prisoner, who had been travelling with Augustus, unaccompanied by servants, put up at an inn near Paris. In the course of the evening Augustus complained of illness, and Castaign prescribed for him sugared wine. Castaign left his companion at four o'clock in the morning, to take, as he said, a walk; but it was proved that he went to Paris, and procured, at a druggist's shop, twelve grains of tartar emetic and half a drachm of acetate of morphia, assigning as a reason for the purchase at this unseasonable time, that he required it for the performance of experiments on dogs and cats.<sup>1</sup> He immediately returned to Ballet, who was still lying ill, and he prescribed for him cold milk, which he himself administered to him. In five minutes Ballet was seized with convulsions, and, in half an hour, with vomiting and purging. A physician was called in, who, on the representation of Castaign, treated the case as cholera

<sup>1</sup> In Palmer's case, there was some attempt at a suggestion of this kind, but it was too improbable to be carried out. Some dogs were said to have worried the prisoner's brood-mares, and for the purpose of poisoning them, it was to be assumed that the prisoner had secretly procured by gift, three grains of strychnia, late on the Monday evening, within three hours of Cook's first attack, and six grains at another place on Tuesday, within twelve hours of the second attack and death! The strychnia so procured, had evidently been disposed of in some way; for of the three poisons purchased on the Tuesday, prussic acid, Battley's sedative solution, and strychnia, the first two were found on the prisoner's premises unopened, but the strychnia had disappeared. It would have been difficult to persuade a jury, that for the purpose of poisoning dogs three grains of strychnia were procured secretly at a late hour on Monday night in November, or that the whole quantity (nine grains) was such as a man seriously intending to destroy dogs would have procured in two separate quantities, under the circumstances. It was clearly impossible to account in a way consistent with innocence for the possession of the poison.

morbus. In a few hours the patient became quite insensible ; he was unable to swallow, was bathed in a cold sweat, with a small pulse, stertorous breathing, a contracted pupil, a hot skin, the jaws locked, the neck rigid, the abdomen tense, and the limbs affected by spasmodic convulsions. In this state he died, about thirty hours after the first symptoms. The only morbid appearances, on inspection, were congestion of the brain, and serous effusion in the cerebral membranes. The stomach and its contents, as well as other parts of the body, were submitted to a very minute and elaborate chemical analysis, by Vauquelin, Lherminier, Magendie, Barruel, Segalas, and Pelletan, comprising the most eminent organic chemists in France. Chaussier and Pelletan attended the investigation on the part of the prisoner. Morphia, strychnia, and brucia, were especially sought for, by processes and tests quite appropriate for use in the present day.<sup>1</sup> The conclusion come to by these chemists was, that there was no trace of any one of these poisons in the body.

The medical defence was, that as no poison was found in the body it was not a case of poisoning. Orfila, in giving his evidence, said that poisons might cause death, and yet not be detected, owing to their removal by vomiting or absorption. Chaussier, who appeared for the defence, was asked whether the acetate of morphia could be detected in the dead body? *Yes, to a molecule.* But when it is absorbed, is it then possible to find it? It requires a long time to be absorbed, and when *the poison cannot be found the corpus delicti is wanting.* Does acetate of morphia produce a dilatation of the pupil? Yes. You do not agree with Dr. Orfila? I have experience that Dr. Orfila does not possess. In spite of the non-detection of poison, however, the jury were quite satisfied that Augustus Ballet had died from acetate of morphia, administered by the prisoner, and he was convicted and executed.

This is not a solitary case. A woman of the name of M<sup>c</sup>Conkey was tried at the Monaghan Lent Assizes, in 1841, for the murder of her husband by poison. The analysis was intrusted to my friend, Dr. Geoghegan, Professor of Medical Jurisprudence in the Royal College of Surgeons, a good chemist, and

<sup>1</sup> The test used for morphia is that which is now employed by toxicologists,—a bitter taste, and the colour produced by nitric acid.



an experienced medical jurist. The stomach, in this case, had not been cut open and its contents mixed with the intestines in one large jar, but they had been carefully and separately packed and sealed. In stating the result, he says: "After a careful chemical examination of the coats of the stomach, its mucus, and the contents of the intestine, the particulars of which it is unnecessary to detail, I was unable to detect any trace of poison." The symptoms, however, were clearly those of poisoning by monkshood or aconite, and the morbid appearances were corroborative. The woman was very properly convicted and executed. Previous to execution, she confessed that her husband had been poisoned by the root of "*blue rocket*" (*aconitum napellus*), which she said had been mixed with pepper by an acquaintance of her's, and sprinkled over some greens, which her husband had eaten at his dinner. She denied, however, any knowledge of the transaction at the time of its occurrence, but this denial was quite inconsistent with moral circumstances. Dr. Geoghegan refers to other instances of general poisoning, in which the fact of poisoning, *in the abstract*, was determined from medical and moral circumstances, without any exact evidence as to the nature of the *special poison* employed; *e.g.* those of Thom, for the murder of the Mitchells, at Aberdeen, 1821; of Mary Ann Alcorn, Edinburgh, 1827; and of Rachel Shannon, tried at the Cork Lent Assizes, 1837.<sup>1</sup> "There can be very little doubt," he observes, "that many additional cases of this kind would have come to light, had medical men devoted more attention to the discrimination of the circumstances under which an opinion in favour of poisoning in the *abstract* may be given, although there be *no chemical indication of the special agent*. It should be borne in mind, that in those instances in which the medical inspector, although unable to give a *decided* opinion, has good grounds for concluding the fact of poisoning to be highly *probable*, it becomes his duty to state this distinctly, when examined, as possibly such an opinion, when conjoined with strong moral evidence, may enable the jury to render the cause of death (constructively) certain."<sup>2</sup>

In the course of some years devoted to toxicological

<sup>1</sup> Jebb's 'Reserved Cases,' Dublin, 1841, p. 209.

<sup>2</sup> See his paper on Poisoning by Monkshood, in the 'Dublin Medical Journal.'

researches, I have met with several instances of poisoning by vegetable, and some by mineral substances, in which no trace of poison could be detected in the body, although the stomach and its contents had been properly preserved. It was not until the occurrence of Palmer's trial, that I had ever heard facts of this kind seriously disputed. It has been hitherto considered that volatile poisons, such as Prussic acid, when taken only in small but fatal doses, are apt to disappear by their volatility, and may no longer be found in the body. It has been supposed that vomiting and purging might get rid of poison,—the person die, and no trace be found in the body. Instances of this I have met with in suicidal deaths from sulphuric and oxalic acids, and in several cases of undoubted poisoning by opium in a liquid state. It has been usual to consider that rapid absorption and elimination might account for the disappearance of some poisons, and that, for this reason, if the dose were small and just sufficient to prove fatal, the chance of finding it would be less than if it were large. Dr. Christison, writing of opium, in the last edition of his work on Poisons, says, "but according to my own observations, the poison *will often disappear in a short time*, so far as to render an analysis abortive. Thus, in the case of a young woman, who died five hours after taking not less than two ounces of laudanum (which would be equivalent to at least fifty grains of opium), I could apply, to the fluid procured from the contents of the stomach, only the test of its taste, which had the bitterness of morphia. In the case of another young woman, whose stomach was emptied by the stomach-pump, four hours after she had taken two ounces of laudanum, I could obtain from the evacuated fluid, when properly prepared, only the indications of the presence of morphia, supplied by its bitterness with the imperfect action of nitric acid, and the indication of the presence of meconic acid supplied by the imperfect action of perchloride of iron. In a third case, where the stomach was evacuated two hours after seven drachms of laudanum (equivalent to at least twenty-one grains of opium), had been swallowed, even the first portions of fluid withdrawn, had not any opiate odour, and did not yield any indication of the presence even of meconic acid. Now, on the one hand, the quantity taken in these instances is rarely exceeded in cases

of poisoning with laudanum; and, on the other hand, the interval during which it remained in the stomach subject to vital operations, is considerably less than the average in medico-legal, and above all in fatal cases. It may be laid down, therefore, as a general rule, that in poisoning with opium, the medical jurist, by the best methods of analysis yet known, will often fail in procuring satisfactory evidence, and sometimes fail to obtain any evidence at all of the existence of poison in the contents of the stomach.”<sup>1</sup> Such is the testimony of an accurate observer regarding the means of “infallibly” detecting in the body a poison which causes annually, in this country, as many deaths as are occasioned by all other poisons taken together. This testimony too is not got up to meet a particular set of circumstances, but it is the result of long and accurate observation.

When the quantity of opium taken is small, and the person survives a few hours, neither the morphia, nor the meconic acid, for which a chemist looks, can be detected. In one case which I had to examine, a child about six years of age, died from three-quarters of a grain of opium given in divided doses. It survived thirty-six hours from the time of commencing the medicine, and had not taken any portion for some hours before it died. On examining the stomach and bowels, there was not the smallest trace of opium, of morphia, or meconic acid to be detected. At the maximum, the quantity of morphia which killed this child, would not have exceeded one tenth of a grain, distributed in doses over thirty-six hours. Infants have been frequently killed in a few hours by doses of opiate preparations corresponding to the fifth, the eighth, the tenth, and even the twelfth part of a grain of opium, equivalent, in the first case, to one fortieth of a grain, and in the last case, to one ninety-sixth of a grain of morphia. Dr. E. Smith, has lately recorded a case in which an infant, seven days old, died comatose eighteen hours after having taken one minim of tincture of opium. Symptoms of narcotic poisoning set in, in about half an hour, and the child never rallied from the effects of the drug. Here not more than one twelfth of a grain of opium, corresponding to the 120th grain

<sup>1</sup> ‘Treatise on Poisons,’ 4th edition, p. 697.



of morphia, destroyed life ('Medical Times and Gazette,' April 15th, 1854, p. 386).

Is it to be supposed, in reference to such cases, that even if there were no absorption and diffusion of the morphia through the body of the child, a chemist could separate this minute quantity of morphia from the stomach? If a man ventured to assert this, no one who knows practically the properties of opium would believe the assertion. The allegation that morphia must in all cases be found, before death is attributed to narcotic poison, would lead to wholesale infanticide, without any chance of proving the perpetration of the crime.

The conclusion, from this inquiry, therefore, is that there are many poisons which may cause death, under circumstances in which they cannot be detected by chemical analysis in the body.

It now remains to be determined, whether the poison strychnia is an exception to the remarks here made, regarding the detection of organic poisons in general.

CAN A PERSON DIE FROM STRYCHNIA, AND NO TRACE OF THAT POISON BE FOUND BY CHEMICAL ANALYSIS IN THE BODY?

The reader will perceive, that the practical answer to this question must consist, not in the application of chemical tests to a substance which we know to be strychnia out of the body, but to our means of separating it from the solids and fluids of the body in a case in which we do not know, but in which we are called upon to prove that it has been taken. This inquiry, in fact, involves:

1. The chemical processes by which the supposed strychnia may be separated from the solids and liquids of the dead body.
2. The chemical tests applied to determine that the substance so separated, is really strychnia and nothing else.
3. The amount of confidence to be placed in the so-called tests, in a case of alleged murder, where the acquittal or conviction of the prisoner will probably turn upon the answer.

I have put the third proposition in the form above given,

because the occurrence of a case involving this question may be easily conceived, and the trustworthiness of the tests can then, and then only, be duly estimated. I must beg the reader to remember that this is not a question of the adulteration of bitter beer, or any ordinary article of food, or any artificial admixture of poison, in which we are seeking for that which we know to be present; it is one, I must assume, involving the life of an individual, and therefore deserving of serious consideration.

Although contrary to usual practice, but in accordance with the wish of the Attorney-General, who was desirous of giving every possible advantage to those who were defending William Palmer, Dr. Rees and I furnished to the prisoner's solicitor the subjoined statement regarding the processes pursued by us for the detection of strychnia and antimony in the body of Cook.<sup>1</sup>

<sup>1</sup> I may here observe, that copies of the medical evidence, including the reasons for the opinions of the witnesses, were placed in the hands of the prisoner's attorney some time before the trial. His counsel, therefore, had full opportunity of anticipating the statements of the witnesses for the prosecution, and framing their questions for cross-examination accordingly. There can, I conceive, be no objection to this course, when scientific questions are likely to be dealt with *bonâ fide* and with a view to public justice, by witnesses consulted for the defence; but even then, the exchange of documents should be reciprocal. I need hardly observe, however, to those who are acquainted with the special pleading which may be brought to bear on scientific questions, to the confusion of the court and jury, that such a practice must be in the end detrimental to the course of justice.

The verdict in Palmer's case certainly shows that no evil attended it on this occasion, but from its exceptional nature, this case can hardly be quoted as favorable to the practice. If carried out to an equal extent in future cases, it must, in my opinion, be destructive of all confidence between those who are consulted and those who consult. There may, it is well known, be a good use and a bad use of such documents thus confidentially furnished. There are cases in which the object of the defence is not any abstract idea of public justice, but *per fas aut nefas*, the "saving of the life" of the person charged with murder. A medical witness who knows that his chemical processes and opinions are to be submitted to the judgment of hostile critics, employed especially for this purpose, may be induced either to decline the responsibility of the investigation altogether, or to place his statements in such a form as to render the prosecution nugatory. If the rule be good for criminal, it will be equally good for civil cases; if good for medicine, it is equally good for law; and there would be no reason why counsel for prosecution and defence should not furnish each other before trial with abstracts of their intended speeches and the points of evidence on which they intend especially to rely, as well as lists of the scientific witnesses whom they intend to call, including those kept back because

## PROCESS FOR DETECTING STRYCHNIA.

1. *Analysis before the Inquest.*—Stomach well washed with cold distilled water, to remove every soluble substance from surface; pyloric half of the stomach reserved, and one half of the washings. The cardiac half, with one half of the washings mixed with alcohol about 840, acidulated with diluted sulphuric acid, and warmed to about 170°, frequently stirred and allowed to digest for twenty-four hours. Liquid filtered off through wet filter; pale straw-colour; acid reaction; not bitter to taste. About one fourth of this liquid (amounting to two fluid ounces) was neutralized by carbonate of potash, and gently evaporated to dryness. The dry extract was treated with warm rectified spirit until exhausted. This was filtered. It formed a pale-coloured liquid. Again tasted by Dr. Rees and myself; no perceptible bitterness. A portion of this liquid was evaporated gently on a wide dial glass on sand, and films of a fine crystalline substance were left on the glass.

Sulphuric acid and powdered bichromate of potash produced, with these films, a brownish discoloration, and subsequently, green oxide of chrome. A portion of the residue, evaporated in another glass, gave a purple-reddish tint with sulphuric acid and bichromate of potash, the colour becoming of a deeper red by exposure.

The effect was very similar to that which we have obtained from small traces of bile with saccharine matter and sulphuric acid. Our conclusion was that there was no chemical evidence of the presence of strychnia. A portion of the drainings of the jar were tested for strychnia by a similar process, but the alcoholic acid extract had no bitter taste, and bichromate of potash and sulphuric acid gave, with the evaporated residue, only green oxide of chrome.

2. *After Inquest.*—Another portion of the alcoholic acid liquid (which had been reserved) was subsequently tested by Graham's process, *i. e.*, by animal charcoal, but the result was not satisfactory.

Dr. Rees made a subsequent trial on the pyloric half of the stomach (by diluted hydrochloric acid warmed, and followed by animal charcoal and subsequent digestion in alcohol). In this, as well as in the preceding trial, bichromate of potash, ferricyanide of potassium, and sulphuric acid were used, but the results were equally unsatisfactory. There was a change of colour on the addition of the tests, but no clear or positive evidence of the presence of strychnia.

## AS TO DETECTION OF ANTIMONY.

The process used was that of Reinsch, *i. e.*, by a mixture of hydrochloric acid and water, producing a deposit on copper foil and copper gauge.

The different organs mentioned in the report yielded only slight traces. An acid

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their opinions were adverse. Even scientific gentlemen who allow themselves to be employed for such purposes as those above indicated, would object to this mode of dealing with their own facts and opinions. In short, it is difficult to understand how the criminal and civil business of the country could be conducted on such a principle. I make these remarks rather in reference to future cases, that medical men who venture to give opinions in questions of alleged poisoning may know in what position they may really stand.



decoction (hydrochloric) of the pyloric half of the stomach yielded, with zinc, a black deposit, allowed to collect for some days, when this was digested in nitro-hydrochloric acid, and evaporated to dryness. On removing the chloride of zinc by water, a white insoluble sediment was obtained, which, when treated by hydrosulphuret of ammonia, acquired a reddish-brown colour. The deposits on copper presented, when recent, the peculiar violet colour of antimony, and gave, when heated, no sublimate of octohedral crystals. They possessed all the characters of antimonial deposits. A portion of the antimony was converted to antimoniate of soda by deflagration with nitrate of soda, and this compound was subsequently tested, and its antimonial nature established by the action of a current of sulphuretted hydrogen on the solution, acidulated with hydrochloric acid.

In our judgment, the chemical evidence of the presence of strychnia should be as clear, distinct, conclusive, and satisfactory, as in the case of arsenic or any other detectable poison; and although we obtained, in this instance, by the use of the tests, certain changes of colour, which an ardent imagination might, I believe, have easily construed into proofs of the presence of strychnia, we declined to take this view, and concluded, from the results above mentioned, that in the parts examined by us, namely, the coats of the stomach and the drainings of the jar, there *was no evidence of the presence of strychnia*. Mr. Sergeant Shee, in his address to the jury, gives to Dr. Rees and myself, credit for having done "all that the science of chemical analysis could enable men to do to detect the poison of strychnia." This observation was of course only intended to influence the jury in relying upon his argument, that "strychnia not having been found in Cook's body, under the circumstances of the case, it never could have been there."

I shall only remark, that although there may be a difference of opinion as to the relative efficacy of the processes employed for detecting strychnia, we had satisfied ourselves by a preliminary trial that strychnia, when in sufficient quantity to justify a medical opinion, might be separated and detected by the processes which we here adopted.

With the exception of the stomach and the drainings of the jar, no analysis for strychnia was made. We knew no process which could be safely relied on for the separation of strychnia from the tissues; and on this ground we directed our analyses of these parts to the detection of antimony and mercury. Some chemists, who have manifested great wisdom

after the event, in reference to this case, would have devoted their entire researches to the detection of strychnia; and they imagine that they could have found it, if not in the stomach and liquids of the jar, at least in the liver, spleen, and kidneys—nay even in the *bones* of Cook! They forget, however, that at this time there was no suspicion of death from strychnia, no knowledge of symptoms to indicate it; and assuming that there was a satisfactory method of obtaining the poison in a dry and tangible form from the liver, there was no more reason for searching the tissues for strychnia than for searching them for morphia, brucia, veratria, picrotoxia, or any other of the numerous alkaloidal poisons.<sup>1</sup> Unless gifted with the power of pre-vision, it is not probable that their skill would have been directed to this special research; and without it they must have subdivided the organs into various portions to search for the numerous poisons which can occasion sudden death. If they had not taken this course they would probably have incurred a charge of carelessness, in neglecting to seek for the very poison which might be subsequently proved to have caused death. Dr. Rees and I had no confidence in the processes

<sup>1</sup> I stated, in answer to questions put by the learned sergeant, that we had no suspicion of strychnia, until the 4th December, *after the analysis was completed*, and that information of the purchase of strychnia was then for the first time made known to us, by a note from Mr. Gardner. Further, this knowledge did not in any way affect our opinion, that the cause of the death of Cook was still unexplained. It was only after reading Jones's statement of the mode in which the deceased died, and hearing his evidence, and that of Mills, that I came to the conclusion that strychnia must have been the poison used. *Ten days* had then elapsed, and yet the learned sergeant was allowed, without comment or contradiction, to put the following statement to the jury. "They (Drs. Taylor and Rees) had *distinct information* from the executor and a near relative of the deceased, either personally or through his solicitor, that *he from some cause or other had reason to suspect the poison of strychnia*; they undertook the examination of the stomach, which I think upon the whole evidence, without adverting to that part of it now in detail, you will be satisfied was not in an unfavorable condition for a sufficiently accurate analysis, *with the expectation, that if strychnia had been taken, it would be found, and without any doubt as to the efficiency of their tests to detect it.*" This statement was, upon the counsel's own questions, quite contrary to the evidence given on oath. Juries are desired to dismiss from their minds all that they have heard before entering the Court, the fear being that they may act upon the belief of something which is unproved and untrue. Of what utility is such a rule as this for guiding them to a verdict, if misrepresentations of scientific evidence are thus allowed to be placed before them.

relied on for the detection of strychnia, morphia, &c., in the tissues; and even had we obtained the results which were considered by the chemists for the defence to be conclusive of the presence of strychnia, we should have declined to base our opinion of the cause of death upon such precarious characters. But in fact, up to that time strychnia had not been discovered in the tissues, to our knowledge, in any fatal case of poisoning by it which had occurred in this country. It had not even been sought for by chemists who had had the opportunity, such as Mr. Herapath. His researches for the poison never appear to have gone beyond the contents of the stomach. We could not find that Orfila, Christison, or any toxicologist of repute, had detected this poison in, and separated it from the tissues in any one instance, either of a human being or of an animal. For these reasons the tissues were not examined, and it was therefore not in our power to say whether strychnia was or was not present in them. We considered that the tests and processes open to us for such a purpose, were too fallacious to allow us to decide the question by resorting to their use. Another course was, however, open to chemists having greater confidence in their chemical processes, who, to use the language of Sergeant Shee in his address to the jury, "were ready to depose on their oaths, that not only if half a grain, or the fiftieth part of a grain, but I believe they will go on to say that if five, ten, or twenty times less (*i. e.* one thousandth of a grain), *had entered into the human frame at all*, it could be and *must be detected by tests which are unerring.*" I need hardly remark, to those who are acquainted with the properties of strychnia, that this is a rash and unfounded statement, calculated to deceive the jury to whom it was addressed, and to lead them to come to a wrong conclusion on the scientific facts. It was entirely unsupported by any evidence which was adduced in the case.

I have elsewhere stated, that the body of Cook was exhumed on the 25th of January, and the solicitor for the prisoner instructed two medical gentlemen to attend on the part of the defence at the exhumation. The fact that strychnia had not been found in the stomach of Cook was then well known to him; and it was in his power to cause portions of the body to be removed for analysis by his chemists, if he was dissatisfied with the results



which had been obtained by Dr. Rees and myself. We had declined to undertake or to rely upon an analysis of the tissues for organic poisons, but there was no impediment to the chemical witnesses, the "higher adepts" in science, employed for the defence, undertaking this investigation. The state of the body on the 25th of January, could not have interfered with their analysis. One of them, Mr. Rodgers, professes to discover with great facility strychnia in the *bones*; and Mr. Herapath deposed on oath, that if this poison had caused death, he could detect it up to the time that the body had become "completely decomposed;" in fact, when it was converted to a "*dry powder*." Nothing could be more positive than this evidence: hence the non-analysis of the residue of Cook's body, when an opportunity was presented, shows that those who were most earnest in defending Palmer, believed that Cook had been poisoned by strychnia, and that it would have been dangerous to intrust the analysis to their own witnesses; to gentlemen, in fact, who professed to detect in the dry pulverulent remains, even the 1000th part of a grain, had it been taken during life. Let us consider the result of such a proceeding, had it been adopted. If the strychnia had been found, and its presence in the body of Cook proved to the satisfaction of the witnesses for the defence, this would have only corroborated the evidence for the prosecution, and sealed the fate of the prisoner. If, on the other hand, they reported that they had not found the poison in the tissues, or that the indications of its presence were not to their minds satisfactory, this would have still left the case for the prosecution to be answered just as it then stood; namely, that the symptoms of Cook could only be ascribed to the effects of strychnia, and the negative results of the analysis would have no more assisted the defence than the negative results already obtained by Dr. Rees and myself.

I may here remark, that various incorrect statements respecting the detection of strychnia in cases of poisoning, have been erroneously attributed to me. It has been represented, that at the inquest on Cook, I had given an opinion to the effect that, after an hour had elapsed, when strychnia had been taken as a poison, it could not be proved to exist in the dead body by chemical tests! My statement was to the effect, that strychnia was not so susceptible of detection after death

as arsenic, and other metallic poisons; that it might destroy life in small doses (*half a grain*);<sup>1</sup> and that, according to circumstances, enough *might be absorbed in an hour*, to kill a person. Further, when thus removed from the stomach by absorption, I knew of no instance in which it had ever been detected in the dead body. This matter was very properly placed before the jury, by the learned Attorney-General, in his opening address :

"Sometimes strychnia is found, at other times it is not. It depends upon circumstances. A most minute dose will destroy life, from half to three quarters of a grain will lay the strongest man prostrate. But in order to produce that fatal effect, it must be absorbed into the system, and the absorption takes place in a shorter or longer period, according to the manner in which the poison is presented to the surfaces with which it comes in contact. If it is in a fluid form, it is rapidly taken up, and soon produces its effects; if not, it requires to be absorbed, and the effects are a longer time in showing themselves. But in either case, there is a difficulty in discovering its presence. If it acts only on the nervous system through the circulation, an almost infinitesimal quantity will be present. And as it is a vegetable poison, the tests which can be employed are infinitely more delicate and difficult than those which are applied to other poisons. It is unlike a mineral poison, which can soon be detected and reproduced. If the dose of strychnia has been large, death may ensue before the whole has been absorbed, and a portion is then left in the stomach or intestines. But if a *minimum* dose has been administered, a different consequence follows, and the whole may be absorbed. Practical experience bears out the theory that I am enunciating. Experiments have been tried, which show, that where the same amount of poison has been administered to animals of the same species, death will ensue in about the same number of minutes, accompanied by precisely the same kind of symptoms; while, in the analysis afterwards made, the presence of poison will be detected in one case and not in another. It has been repeated over and over again, that the scientific men employed in this case, had come to the conclusion that *the presence of strychnia could not be detected by any tests known to science*. They have been grievously misunderstood. *They never made any such assertion*. What they have asserted is this—the detection of its presence, where its administration is a matter of certainty, is a matter of the greatest uncertainty. It would, indeed, be a fatal thing to sanction the notion that strychnia administered for the purpose of taking away life, cannot afterwards be detected! Lamentable enough is the uncertainty of detection! Happily, Providence, which has placed this fatal agent at the disposition of man, has marked its effects *with characteristic symptoms*, distinguishable from those of all other agents by the eye of science."

These are the words of truth, and an accurate repre-

<sup>1</sup> See in the Table, p. 349, Case No. 9, that of Dr. Warner, who died in twenty minutes from a dose corresponding to less than half a grain of pure strychnia.

sentation of the present state of knowledge in reference to this subject.<sup>1</sup>

The principal reasons for the non-detection of strychnia in the body of a person who has died from its effects may depend on—

1. *The quantity taken.* If the dose be small, from one half to three quarters of a grain, it may be rapidly absorbed and removed from the stomach. It is only the *surplus* of a fatal dose which is found in the stomach after death. If a man swallows a dose of ten, fifteen, or twenty grains, and dies quickly, without vomiting, then the residue, or some portion of it, may be found.

<sup>1</sup> In a letter addressed to Sir George Grey, dated March 17th, 1856, Mr. John Smith, solicitor for the prisoner, thus expresses himself:—"Allow me to call your attention to the Leeds case of poisoning by strychnia, where that poison was found, whereas Dr. Taylor *swore it was not traceable.*" I need scarcely observe, that this is an untruthful representation of what I did state, at the inquest on Cook, at which Mr. Smith was not present, and therefore could not of his own knowledge know what I did say. I stated that, *in Cook's case, the poison was not to be detected*, and that a person might in some cases die from this poison without any of it being found in the body; but I did not say that it never could be traced in any case, or under any circumstances. The comparison of the Leeds with Cook's case was manifestly unfair. Mrs. Dove had been dosed with strychnia, at intervals, for a *week* before her death. The last and fatal dose, obviously a large one, was given *diffused in liquid*, only *twenty minutes* before she died. The medical gentlemen who made the analysis (one of them at least) had a full knowledge of the symptoms from personal observation, and *suspected strychnia before her death.* Their analysis was therefore directed exclusively to this poison. They themselves removed the stomach and secured the contents, and after applying to them various tests and processes during several days, had, it appears, still enough of the poison left in the extract of the stomach to kill four animals! Is there any one feature in which the analyses made in the cases of Cook and Dove, can be placed in comparison, or any fact to justify the untruthful statement contained in the letter to Sir George Grey. A medical witness, however, must always be prepared for an ample amount of misrepresentation when the sole object of a solicitor is "to save the life" of his client. The lives of those who have perished by poison are altogether put out of consideration. This appears to be a grievous wrong without any remedy. A solicitor engaged in a case of this kind has an unbounded license extended to him. If the witness is silent under such false imputations, the class of men who are likely to form the jury will think that he is wrong, and has committed a grave error; if, on the other hand, he publishes a contradiction to such untruths, he is immediately charged with prejudice against the "unhappy" prisoner, who is represented as never having written anything against him! To the circulation of such false statements may, in some measure, be traced the crime of Dove in poisoning his wife at Leeds.



2. *On the time which has elapsed after taking the strychnia, until the symptoms commence.* The longer this interval, the greater the quantity of poison removed from the stomach by absorption. The poison has been found to be diffused through the circulation in nine minutes. If the person dies in ten or twenty minutes from the time of swallowing the strychnia, some may be found. If he lives an hour or longer, the greater portion may be removed by absorption.

3. *On the careful preservation of the stomach and its contents.* If the fluid or solid remaining in the stomach at the time of death is not carefully preserved, there is a great probability, if the residuary quantity be small, that it will not be found.

In testing the case of Cook by these propositions, we may remark—

1. The doses were probably small: on Monday night the quantity was not sufficient to prove fatal. On Monday and Tuesday nights a long period elapsed on each occasion before the symptoms commenced. This is not in accordance with the taking of a very large dose. As a medical man, the prisoner knew well the dose which might suffice to kill; and, after the experience acquired by him of the effects of strychnia on Cook on Monday night, he could so adjust the dose as to destroy him without incurring the risk of leaving any great amount of residue. A dose of from three quarters of a grain to a grain would have sufficed to produce the effects on Tuesday night.

2. An hour and a quarter had elapsed before any symptoms manifested themselves in Cook. If absorption commences in a few minutes, and continues throughout, there was time for the removal of the greater part by this process during the hour and a half that he survived.

3. The stomach of deceased was delivered to us in a condition most unfit for analysis, or for the detection of such a poison as strychnia. I am informed that it was accidentally cut during the examination of the body, and some of the contents had escaped. If put into the jar in the mode described by the witnesses in evidence, it is perfectly clear that there must

have been some tampering with it subsequently.<sup>1</sup> When it reached us, it was laid open from end to end, and *turned inside out*. No person making an inspection for the purposes of a chemical analysis, with the slightest knowledge of his duty, could be guilty of an act of this kind. If he had unfortunately lost the contents, wholly or in part, during the examination, as a rational person he would have preserved as much of the stomach as it was in his power to do; or would have placed it in the jar with the ends secured, and in a different condition from that in which we found it.

The fact that the poison was given to Cook in the form of pill was unfavorable to the adhesion of strychnia to the stomach; since, until the structure of the pill had been broken up, the crystals or powder could not come in contact with the mucous membrane; but it is not necessary that the structure of the pill should be entirely broken up, in order that the poison should operate fatally. A partial solution from the outside would suffice. Since this trial, the results of two experiments, performed by persons independently of each other, one by Mr. Devonshire, a witness in this case, and the other by Mr. Horsley, of Cheltenham, have been communicated to me. In each instance a dose of strychnia, closely wrapped in paper, was administered to animals; in one, a grain was given to a cat, in the other, two grains to a dog. After death the strychnia (in the cat) was found in the stomach still in the paper wrapper,—the quantity apparently undiminished. The animals must have been poisoned by the imbibition and solution of a minute portion of strychnia through the paper. Assuming that any part of the pill remained in Cook's stomach at the time of death,—as a cut was made into the greater curvature during the inspection, it is possible that the residuary portion of pill may have escaped from the aperture unobserved with some of the fluid. Of course this is a mere speculation; but it would account for the loss of strychnia from the stomach. My belief is, that in Cook's case a small dose (but sufficient to kill) was given—that during the hour and a half which he survived after swallowing the poison, some portion (sufficient to account for death) was carried into the body by absorption, and that the residue through inadvertence, was lost during the inspection. I am

<sup>1</sup> See p. 278, *ante*.

informed by a gentleman who assisted at the inspection, that he had at the time no suspicion of poison, or more care would certainly have been taken.

The non-discovery of strychnia in the coats of Cook's stomach is therefore no great mystery; it was not owing to the failure of chemical tests, the influence of tartar emetic, putrefaction, eremacausis, or any of those occult causes which have been made the subject of newspaper speculation, but simply to the absence of the poison from that part of the body in which, if present in sufficient quantity, a chemist may have a reasonable expectation of finding it. The test of the accuracy of this view will be not in poisoning animals with doses of strychnia six times as great in proportion as the dose which would have sufficed to kill Cook, not in carefully removing the stomach without losing the contents—not in directing the entire analysis to strychnia alone, with a full knowledge that it has been taken and with a full impression that it must and will be found somewhere,—but, in the first case of a human being who may be unfortunately poisoned by a grain or a grain and a half of strychnia after it has remained an hour or longer in the living body,—in conducting the inspection as it was conducted in Cook's case, cutting the stomach from one end to the other, losing part of the contents and diffusing the remainder in a large jar containing blood, fæces and the whole of the intestinal canal. Let the viscera thus treated be placed, without any information being given, in the hands of an "adept" who professes to be able to detect the minutest fractional quantities of strychnia in a dead body, and we may then judge how far his chemical tests will bear out his statement. It is not improbable that on the discovery of one poison (antimony), diffused throughout the body, he would stop, and not direct his analysis to the research for any other.<sup>1</sup>

<sup>1</sup> The presence of poisons may be occasionally overlooked, even by chemists of great repute as analysts. In the case of *Stephens v. Barwell*, tried at the Wells Autumn Assizes, 1855, Mr. Herapath gave evidence for the plaintiff, on the composition of a certain slag found on the plaintiff's ground. He had found in it lead, zinc, silica, &c.; but when asked by Mr. Montague Smith, whether he had discovered *arsenic*, he stated that he had not looked for it. Arsenic was found in the slag by Mr. Brande, Mr. Johnson, the assayer, and myself, and its presence there had rather an important and adverse bearing on the plaintiff's case.



With this statement of the facts which have excited so much anxiety in the public mind, let the reader consider the form in which the counsel for the defence was permitted to address the jury on this important question :

"If he (John Parsons Cook) died from the poison of strychnia, he died within two hours of the administration to him of a *very strong dose*<sup>1</sup> of it,—he died within a quarter of an hour or twenty minutes of the effects of that dose being visible in the convulsions of his body : the post-mortem examination took place within six days of his death,—there is not the least reason to suppose that between the time of the injection of the poison, if poison was taken, and the paroxysm in which he died, there was any dilution of it in the stomach, or any ejection of it by vomiting. *Never therefore were circumstances more favorable ; unless the science of chemical analysis is altogether a failure for the detection of the poison of strychnia, never was there a case in which it ought to have been so easy to produce it.*"<sup>2</sup>

One other point here requires to be adverted to, since it formed a conspicuous portion of the learned counsel's defence :

"The gentlemen who have come to the conclusion that strychnia may have been *there* (*i. e.* in Mr. Cook's *body*, more correctly his stomach), have arrived at that conclusion by

<sup>1</sup> There is not the slightest foundation for this statement. If Cook had taken a "very strong dose," the symptoms would not probably have been so long protracted as an hour or upwards, but would have appeared in from fifteen to twenty minutes. Further, although the term "very strong" has no definite meaning, a large quantity of strychnia could not have been contained in two small pills, such as were given by the prisoner to the deceased. The jury, however, were to be impressed with the idea that Cook's stomach was saturated with strychnia, and therefore it ought not to have escaped the processes of the chemists, especially as the cutting up of the stomach, and the diffusion of its contents somewhere, were described to be circumstances most favorable to the analysis!

<sup>2</sup> In the case of Wooler, tried at the Durham Winter Assizes, 1855, another learned sergeant, in conducting the defence, argued strongly in favour of the innocence of the accused, on the ground that having strychnia in his possession, he would have used strychnia and not arsenic (the poison found in the body, and of which the deceased died), because "it is now pretty universally known that there is no poison so certainly detected after death as arsenic," while to a man skilled in drugs, it was known "that there was no poison so difficult of detection and discovery as that of strychnia."

experiments of a very partial kind indeed; they contend that the poison of strychnia is of that nature, that when once it has done its fatal work and become absorbed into the system, it ceases to be the thing which it was when taken into the system; it becomes decomposed, its elements separated from each other, and therefore no longer capable of responding to the tests which, according to them, would certainly (?) detect the poison of undecomposed strychnia: that is their case. They account for the fact that it was not found, and for their still retaining the belief that it destroyed Mr. Cook, by that hypothesis."

Further, Mr. John Smith, the solicitor, in addressing Sir George Grey, with a view to a respite of the prisoner after his conviction, raised a plea upon the same ground,—“the discrepancy of the medical testimony as to the power of finding strychnia.” He observes:—

“Dr. Letheby and Mr. Herapath, two of the most eminent toxicologists of this day, upon *their solemn oaths* declared that they could discover the *fifty thousandth part* of a grain of strychnia, and that if Cook died from that poison they could now find it. Their opinions were confirmed by some of the most distinguished members of the Schools of London (?), Leeds, Edinburgh (?), and Dublin (?). Yet the body (more correctly the coats of the stomach) of Cook did not yield to the manipulations of Drs. Taylor and Rees, the smallest particle of strychnia. Since the unfortunate termination of the trial, my table has been laden with communications of scientific men (?), in confirmation of Dr. Letheby and others.”

Whether strychnia can always be found in the body of a person poisoned by it, if there be only the 1-50,000th or even the 1-1000th of a grain or less, diffused through it, is a question of fact, and not at all dependent on any theory. The extensive experience on which the answer to this question has been so conclusively settled in the affirmative by the counsel and solicitor for the prisoner, in reliance on their witnesses, will be presently examined. If a person or an animal can die from a dose of strychnia, which will leave no clear indication of its presence to the so-called chemical tests, it is practically quite unnecessary to discuss in a Court of law what becomes of

the strychnia, whether it circulates through the blood in a state as permanent and unchangeable as silica (flint) or iron, or whether it becomes decomposed and its elements separated or so combined with the animal structures as, at least in some cases, to be withdrawn from the action of chemical tests. Dr. Rees and I adopted the opinion of Liebig, who was not called for the defence (as it was previously announced he would be), because on this point, he would have probably been a most inconvenient witness to the learned counsel. The theory of the partial, not (as alleged) the complete, metamorphosis of alkaloids, such as strychnia, in the blood, is Liebig's, and not ours: but such a question really could not be imported into Cook's case without a complete disregard of all the facts proved in the scientific evidence. In the first place, the substance of the stomach, and not the body of Cook, was examined for strychnia. It was distinctly stated, in answer to an inquiry from the learned judge, that the organs in which Sergeant Shee thought the strychnia had been decomposed were not submitted to any analysis for strychnia. Hence to argue on a theoretical cause for the disappearance of strychnia from the body of Cook, when there was no evidence to show whether it was or was not present in the part to which it would be carried by the blood, was a palpable absurdity. It had the intended effect, however, of temporarily withdrawing the attention of the jury from the real question at issue.

There is another aspect in which this question may be viewed. Certain results have been elsewhere (*ante*, p. 328) described as having been obtained by us from the application of the tests for strychnia to the evaporated residue from the stomach of Cook. I will assume that Dr. Rees and I had taken a more sanguine view of this matter than we did, and that after hearing of the possession of strychnia by the prisoner, and reading Jones's statement, we had attached greater importance to these chemical results, and had represented them to be indicative of the presence of this poison in the body, what then would have been the effect of our evidence? The learned counsel for the defence would no doubt have addressed the jury somewhat in this form:

"Gentlemen,—I throw aside symptoms which no doubt you will agree with me,



“ as the evidence among the medical witnesses is conflicting, may have been due to  
“ disease and not to poison, and I will at once grapple with the main, I may say the  
“ vital question, in this most important case. We are told that strychnia has been  
“ detected in the body of Cook, and two witnesses for the Crown have been called,  
“ gentlemen, I admit, of great respectability, who have sworn on their solemn oaths  
“ that they found a quantity of this subtle poison, equivalent to the thousandth of a  
“ grain, in the liver of the deceased, and the one hundredth of a grain in the coats  
“ of his stomach. I endeavoured to procure from them, in cross-examination, some  
“ kind of information respecting the mode in which they arrived at this conclusion,  
“ and what did it turn out to be? Why, gentlemen, you heard what they said—a  
“ blue, purple, and red colour, upon adding to some sort of extract obtained from the  
“ dead body of this wretched man, a mixture of bichromate of potash and oil of vitriol.  
“ I am obliged to my friend Mr. Grove, who is himself an accomplished chemist, for  
“ reminding me that bichromate of potash is a strongly coloured substance, and  
“ actually derives its name from the large variety of coloured compounds which it  
“ produces. So he tells me, and I have no reason to doubt his word. Oil of vitriol  
“ I am told, also produces colours with a great number of substances of an organic  
“ nature. Gentlemen, I have a great respect for science, but when I find two wit-  
“ nesses thus coming forward to swear away a man's life upon the hundredth, aye,  
“ the thousandth of a grain of something which they suppose to be strychnia, just  
“ because they noticed a little flickering blue and purple colour when they added  
“ their chemicals to it, I cannot suppress my indignation. Then again they tell us,  
“ it is true, they took extraordinary precautions to get rid of the organic matter.  
“ What, I should like to know, is strychnia but organic matter? and yet they wish  
“ to persuade you and me that they can get entirely rid of one kind of organic  
“ matter and preserve the other kind—the invisible thousandth of a grain—in a per-  
“ fectly pure state, for the production of their blue and purple colours. Gentlemen,  
“ I am almost ashamed to dwell on this part of the case for the Crown. What! a  
“ man's life to depend on the alleged detection of the thousandth of a grain of  
“ strychnia, and that, as I understand, not actually separated in a tangible form, but  
“ merely judged to be present by two learned gentlemen who were diligently looking  
“ for it, expecting it, nay, wishing it, in order to bolster up their theory of the cause  
“ of death: it is a monstrous absurdity, and a part of the evidence of my learned  
“ friend that I am sure your common sense will lead you at once to reject. But I  
“ have hardly put the case of these learned doctors fairly. One of them tells us, as  
“ some foundation for his opinion, that he could discover the fifty thousandth part  
“ of a grain, that is, unmixed with organic matter. I endeavoured to fix him to  
“ some definite quantity which he could detect when mixed with organic matter;  
“ how much he could find in a man's heart, for instance, who had been poisoned by  
“ a grain; because I need hardly tell you, gentlemen of the jury, that the question  
“ which we have here to solve, is the discovery of strychnia in Cook's body, which  
“ may be taken to consist of organic matter. You observed the evasiveness of his  
“ replies: he could not tell—it would depend on the nature of the matter—a small  
“ portion—a very small portion—he had no idea: and it is upon results of this  
“ doubtful kind that these witnesses for the Crown have had the incredible impru-  
“ dence—an imprudence which has led to all this dreadful excitement—an impru-  
“ dence which has rendered it necessary that this inquiry should take place in this  
“ form and in this place, if at all—to state, upon their solemn oaths, that they found

“strychnia in Cook’s body, and that Cook was poisoned by it. I need hardly tell you that, but for this alleged discovery of strychnia in the body, there would be no case against the prisoner.

“But let us deal with this subject seriously. I have been long enough at the bar to remember that gentlemen of equal skill with those who have appeared for the Crown in this case, formerly relied in an equally positive manner on colour-tests for the detection of arsenic. About thirty or forty years ago, whenever a yellow or a green colour was produced by the addition of some chemical tests, the names of which I now forget, to the liquids of a stomach, it was immediately concluded that arsenic was there, and that the person had been poisoned; and some serious mistakes were made in consequence. I am reminded that there have been numerous false charges of poisoning by arsenic, owing to too great a reliance being placed upon the colour-tests for this poison. And let me ask you to consider how the case stands here with respect to strychnia, which the witnesses pretend to have found in this extremely minute quantity in Cook’s body. On what large amount of experience do you suppose these gentlemen to found their very positive statement of the presence of strychnia in this case? Why, one tells us that he has been occupied thirty or forty years as a chemist and toxicologist, and that he has had only one case during that long time, in which he analysed the contents of the stomach and found strychnia. That solitary case, gentlemen, if I understood him correctly, was a case of suicide where a very large dose of strychnia was known to have been taken by the deceased; and where, to help him in his analysis, besides information given to him by the executors and a near relative of the deceased, some of the strychnia was actually found wrapped in paper in the room, or taken from the dead man’s waistcoat pocket, I forget which. The other tells you he has been engaged in these researches for a period of fourteen years, but he has not seen a case of death from strychnia, and therefore has never made an analysis for the discovery of this poison in the body of a person who has died from it. It is upon such extensive experience as this, on the first trial for murder by strychnia which has taken place in this country, that we have two witnesses swearing in the most positive manner that they detected, by the use of tests, which they pretend to describe as unerring, the hundredth part of a grain in Cook’s stomach, and the thousandth part of a grain in his liver; and is it, let me ask you solemnly and seriously, upon evidence such as this that the unfortunate prisoner at the bar is to be sent to the scaffold? Gentlemen, I am inclined to think that their opinions, unsupported by the opinions of others, cannot have much weight with you. However, what I have to say now upon that point is, that I will call before you many gentlemen of the highest eminence in their profession, analytical chemists, to state to you their utter renunciation of the colour-tests, as they have been used by these chemists in their homœopathic analyses. But, gentlemen, assuming for one moment that the witnesses for the Crown are right, and that the substance which they found in Cook’s body was really strychnia, of what value, let me ask, is their evidence? They both admitted to me in cross-examination,—they could not deny it, that the quantity of strychnia which they found in the liver and stomach, taken together, was not sufficient to cause death; and I am prepared to contend, notwithstanding what these men of science tell us, and my witnesses will bear me out in the statement, that unless you discover a sufficient quantity of poison in a dead body to account for death, chemical

“analysis is altogether a failure, and it is the height of rashness to assign death to the poison. Do not misunderstand me, gentlemen; I will make an appeal to your common sense. How can a man die unless there is poison enough in his body to kill him? Talk of the thousandth and the fifty thousandth part of a grain; how could this do injury to a person, when a much larger quantity is given for a medicinal dose, and we are told by these learned witnesses that less than half a grain of strychnia has never been known to destroy life? Besides, admitting it to be strychnia, their science does not even allow them to tell you how long it has been there—whether days, weeks, or months—and, for anything they or you or I know, it may have been the result of the judicious or injudicious use of strychnia at some former period of this unfortunate young man’s career; in fact, long before he had ever seen or heard of the prisoner at the bar. I have already had to comment on the question of the antimony found in Cook’s body. I attached no importance to that, because half a grain, the quantity found, is admitted to be really harmless, and the evidence is, therefore, not of the slightest importance in the case. If we reject the half grain of antimony as unimportant, how can we attach any importance to the presence of the hundredth or of the thousandth of a grain of strychnia. The witnesses for the Crown pretend to account for the small quantity found in the body by the effect of vomiting, absorption, decomposition in the blood, elimination, and other fine theories which no one but themselves can understand. But, gentlemen, if Cook was poisoned at all, he must have been poisoned by a very strong dose—many grains; and can you suppose that in the short space of two hours there would have been no more strychnia left in his stomach and liver than these chemists profess to have discovered? The gentlemen I intend to call will tell you that such theories as these, which they have heard for the first time in this Court, are utterly unworthy of credence. Away then with the colour-tests; let the witnesses produce from Cook’s body a quantity of strychnia which will alone satisfy men of your intelligence that it was sufficient to cause death,—let them produce at least half a grain of strychnia, or their chemical tests are of no value to us. In a memorable case of poisoning by prussic acid, which was the subject of a trial for murder some years since, one grain of this poison was stated to have been found in the stomach; and it was very properly contended by scientific men engaged for the defence, that, as up to that time no instance had occurred of a person having died from so small a dose, the deceased could not have died from it. Gentlemen, if a grain of this powerful poison may be found in a dead body, and persons of high scientific attainments assure you, upon their solemn oaths, that they attach no importance to it, because the quantity is or was insufficient to cause death, how can you for one moment attach any importance to the evidence of chemical witnesses who, for the first time in their lives, profess to extract their thousandths and fifty thousandths of a grain of strychnia from the solids and fluids of a dead body.”

I entertain no doubt that had Dr. Rees and I relied upon such precarious results as the colour-tests gave to us in this case, this would have been the kind of defence; and objections would have been very properly taken to our evidence. There is no reason why one who has sufficient reliance upon his



chemical processes should not swear to the presence of a thousandth or a fifty thousandth of a grain of strychnia in a dead body; different degrees of evidence satisfy different minds, and we felt that we had the right to exercise that independent judgment which we should have conceded to others in a similar case. That judgment we exercised in excluding altogether results which did not appear to us to be satisfactory on a question so momentous as this. Had we taken a different course, there is not the least doubt that the strong part of the case, namely, the evidence from symptoms, would have been thrown aside, and a direct and successful attack made upon the chemical evidence. Had the results been even much more marked than they were, this evidence could not in my opinion have maintained its ground against the objections that might have been fairly urged against it. The strength of evidence in Cook's case consisted, medically speaking, in the nature, progress, and duration of the symptoms. Whether the chemical evidence would or would not have been stronger had the stomach been sent in a proper and unutilated state, is entirely another question,—one which is now beyond the reach of solution.

When so much stress was laid at this trial upon the facility of detecting strychnia in cases of poisoning by it, it becomes desirable to examine this question by reference to past experience, and the reader will probably expect to find, from the very positive statements made by the chemists for the defence, that there is already a large accumulation of facts under this head in the human subject. But it is not so. Deaths from strychnia have hitherto been comparatively rare in this country, and the cases in which the poison has been found in the stomach, prior to the occurrence of the case of Cook, very few. Of its discovery in the absorbed state,—in the blood, or deposited in the organs, there was not, up to the time of the occurrence of this case, a single instance reported. The principal medico-legal journal of France, the '*Annales d'Hygiène Publique et de Médecine Legale*,' which records all cases of poisoning of interest that occur in that country,—does not contain the report of any instance of poisoning by strychnia since its establishment in 1829, during a period of twenty-seven years. In Henke's '*Zeitschrift für*

die Staatsarzneikunde' there is an equal dearth of facts,—I cannot find that one case has been reported during a period of eighteen years. In Casper's 'Wochenschrift für die gesammte Heilkunde' there is a report of a case which occurred in 1846. The details of this case are given in No. 7 in the subjoined list. Our own British journals, extending over a series of years, have contained the reports of but very few cases. It is possible that some may have been overlooked ; but all that I have been able to find by diligent search or by communication with medical friends are described in the table. There have been many instances in hospital and private practice, in which symptoms of poisoning have followed the accidental administration of strychnia prescribed medicinally ; but as the patients recovered, these will not assist us in the present inquiry. The case No. 10 in the table led to a trial for manslaughter. So far as it is known, there have been three murders by strychnia in this country since its introduction about the year 1820. I allude to the cases of Miss Abercromby in 1830, of J. P. Cook in November 1855, and of Mrs. Dove in March 1856. I have also the particulars of three cases which have given rise to trials for murder in the United States and Canada.

The reader will understand, therefore, that in the subjoined table, the cases are limited to those in which strychnia has destroyed life, and in which the facts are recorded in an authentic form. The *symptoms* and *appearances* in each case are tabulated so as to admit of comparison. It will be instructive to compare these with the objections taken in the defence of William Palmer by some of the medical witnesses. It will be perceived, as I have elsewhere had occasion to state, that the circumstances on which reliance was placed to show that Cook had *not* died from strychnia, have presented themselves repeatedly in cases in which death from strychnia could not be disputed. The publication of this table may be of great value as a guide to medical witnesses in future cases.

TABLE OF CASES OF POISONING BY STRYCHNIA.

No.	Authority and Date of Occurrence.	Dose taken and Symptoms observed. Period of Death.	Post-mortem Appearances.	Analysis and Results.
1	Case in Hospital practice, 1831, Dr. Booth and Dr. Bardsley. 'Trans. Prov. Assoc.,' 1834, ii, 215; 'Med. Times and Gazette,' July, 1856. A man, æt. 46.	<i>One grain and a half</i> taken at a dose, after repeated smaller medicinal doses. Stupor; loss of speech; tetanic convulsions of the whole muscular system; opisthotonos so severe, that but for assistants, patient would have been thrown off the bed; consciousness retained; breathing short, laborious, stertorous; pupils dilated; insensible; slight intermissions of convulsions; tetanic spasms more and more violent; gasping breathing; body rigidly and permanently bent backwards; cold sweats. Death in <i>two hours</i> and <i>three quarters</i> after having taken the poison.	Seven hours after death. Fingers firmly contracted; muscular system generally rigid; blood on dividing scalp; vessels of dura mater turgid with dark-coloured blood; arachnoid opaque, and thickened. In right corpus striatum, an apoplectic clot; brain softened around; serous effusion in ventricles; other parts of brain healthy; spinal membranes highly vascular; pia mater had a florid redness, and congested with arterial blood; four patches of extravasated blood between this membrane and arachnoid, opposite last dorsal and upper lumbar vertebræ; spinal marrow healthy; chest and abdomen not examined.	No analysis.
2	Case recorded by Dr. Blumhardt, reported in Wibmer's 'Arzneimittellehre,' art. "Strychnia," p. 254, 1839. Also 'Med. Corr. Blatt des Wurtemb. Vereins,' 1837; and 'Lancet,' Jan. 7, 1838. A young man, æt. 17	<i>Forty grains</i> (2 scrupel reines strychnin) of <i>pure strychnia</i> taken in a glass of water. Soon began to feel great anxiety and restlessness. Four grains of tartar emetic given produced very little effect. In <i>fifteen minutes</i> after taking the poison, he was stretched on his back, on the bed; countenance escaped;	Inspection made twenty hours after death. In spite of great heat of weather, body unusually stiff and rigid; analysis was made of the contents of the stomach, with a view of determining whether any part of the poison could be chemically demonstrated to be present; <i>but there was</i>	As so large a dose of strychnia had been taken, an analysis was made of the contents of the stomach, with a view of determining whether any part of the poison could be chemically demonstrated to be present; <i>but there was</i>



<p>anxious; head and body stiff. The branches congested; spinal marrow at <i>not a trace of strychnia to be</i> wished to turn on his right side, but the upper part soft, at the lower, hard; <i>found</i>. The blood collected could not, having no power, except general congestion of the brain and its retained its thick consistency over the upper extremities. Had his membranes, with dark fluid blood; sistency, and did not undergo full consciousness, and spoke in a loud cerebellum softer than natural. Viscera putrefaction. It was exatone, in his usual manner, on his con- of abdomen and thorax deficient in mined for strychnia, but no trace was found.</p> <p>dition, occasionally interrupted by a slight stiffness of the jaw; blood, as if this fluid had been forced into the central organs of the nervous breathing became difficult; pulse small and quick; occasional fits of into the central organs of the nervous spasm, involving the jaw and the whole body. With a violent shud- system. Lungs healthy, distended with air, and bloodless; heart dering of all the muscles of his body, became as stiff and rigid as a flabby, and <i>empty</i>, as well as the great vessels near it, rendering statue; arms spasmodically bent at the elbows, and drawn over the it difficult to collect any for chemical examination. The blood every- chest; feet distorted, and soles inclined to each other; retention where fluid. The stomach was full of fresh, solid food; no appear- of consciousness; face and lips lived; eyes protruding, pupils dilated; ance of the poison; mucous membrane about the cardia reddened. moaning; intervals of relaxation. Crushed the tube of stomach. The liver contained more blood than the other abdominal viscera. pump between his teeth. Death in <i>one hour and a half</i> after taking Gall-bladder empty.</p>	<p>Case by Mr. Bennett, occur- <i>One grain and a half of strychnia</i>, taken in solution, on an empty stomach. Symptoms began by twitchings, rather more than <i>an hour after taking the poison</i>; general tremors; limbs rigid; whole body stiffened and straightened; neck drawn back; eyes protruding; pulse imperceptible; face livid; froth from mouth; violent tetanic convulsions, with opisthotonos; hands clenched; arms bent; legs and body ex- tended. She died in a violent fit, <i>two hours and a half</i> after taking the poison.</p>	<p>No analysis.</p>
<p>3</p>	<p>Dr. Watson, Glasgow, Sep- <i>Three quarters of a grain of strychnia</i>. Symptoms came on suddenly in twenty minutes; arms extended and rigid; face flushed; lips livid; jaw not fixed; body curved, opisthotonos. Death in three quarters of an hour from commencement of symptoms; al- together a little over an hour.</p>	<p>No analysis.</p>

No.	Authority and Date of Occurrence.	Dose taken and Symptoms observed. Period of Death.	Post-mortem Appearances.	Analysis and Results.
5	J. C. Evans, November 17, 1845. 'Medical Gazette,' vol. xxxvii, 1846, p. 925. A man, æt. 26.	Dose and symptoms unknown. Found dead. Body and extremities quite stiff; nails imbedded in palms of hands; pupils dilated; eyeballs protruded; dark frothy saliva from mouth; teeth firmly closed. Glass with white powder near.	Thirty-six hours after death. Vessels of head turgid; brain and membranes healthy; lungs healthy, but gorged; heart filled with coagulated blood; stomach contained a dark greenish-looking substance like coffee grounds.	Stomach and contents boiled in water; liquid evaporated, very bitter, milky looking, but cleared by ammonia. <i>Sulphuric acid gave a beautiful rose-pink colour.</i>
6	January, 1846. Cornack's 'Monthly Journal,' Feb., 1846, p. 141; 'Medical Gazette,' vol. xxxvii, p. 254. A woman, æt. 35.	<i>Three grains of strychnia</i> , taken in powder in tea. A flow of saliva; no complaint of taste. In twenty minutes, convulsions, every limb shaking; short intervals of recovery, in which she uttered exclamations expressive of great pain; the least motion produced another paroxysm; shaking so powerful that it required several persons to hold her. Death, <i>one hour and three quarters</i> after taking the powder.	No inspection.	No analysis.
7	Case of a Medical Student, reported by Dr. Theinhardt. Casper's 'Wochen-schrift,' February 28, 1846, p. 143.	About <i>thirty grains of strychnia</i> , taken in spirit; seen in one quarter of an hour afterwards; breathing, pulse, and appearance natural. Spasms in the muscles soon commenced, with hurried respiration; twitchings, followed by perfect rigidity of the whole body. An emetic given without effect. In a few minutes, another paroxysm, with violent motions of the whole body, and opisthotonos. A third and fourth paroxysm followed, the patient groaning and screaming. He died in <i>half</i>	Tongue, gums, and lips, violet-coloured, as well as the fingers and toes; the hands were clenched, and the toes drawn backwards; the whole body stiff and hard, like a piece of wood, bent somewhat backwards. No inspection.	No analysis.

an hour. Had evidently suffered great pain; had been heard to cry out before Dr. Theinhardt's arrival.			
8 Dr. Edwards, 'Edinburgh Monthly Journal,' No. 64, April, 1846, p. 230. An adult man.	Dose and symptoms unknown. Body lying on back, in bed, rigid, and stretched out to its full extent; eyeballs prominent; pupils dilated; mouth firmly shut; arms lying over chest; hands firmly clenched; lower extremities particularly rigid, and left foot concave.	Twenty-eight hours after death. Decomposition commenced; partial relaxation of joints. Brain and membranes healthy, but general turgescence of vessels; lungs congested with dark an hour in vinegar, and filled with fluid blood; some tubercular disease; heart dilated, but with vessels healthy, white precipitate; contained a quantity of dark fluid to pale red by nitric acid; blood; spinal marrow not examined; stomach, intestines, and other viscera healthy.	A bottle labelled poison, and containing strychnia, found near the body. Stomach and contents boiled half an hour in vinegar, and filtered: ammonia gave a fine white precipitate; converted by sesquichloride of iron a pale green; tincture of galls an adhesive precipitate. From these experiments concluded to be strychnia.
9 Case of Dr. Warner, October 11, 1846. 'British American Journal,' August, 1847. An adult, æt. 39.	Half a grain of sulphate of strychnia, taken by mistake for sulph. morphia. Symptoms began in less than five minutes; constriction of throat; tightness of chest; rigidity of muscles, in attempting to move; first complained of want of air, and requested the windows to be raised; violent tetanic convulsions, opisthotonos, lasted five minutes, succeeded by partial calm; livid countenance; frothy matter from mouth; moans; attempts to vomit. Death in from fourteen to twenty minutes; mind clear until the last. <sup>1</sup>	No report of examination of body.	No analysis.

<sup>1</sup> Dr. Warner's case is usually considered to represent the smallest dose of strychnia which has proved fatal. A case occurred in a London hospital, in October, 1853, in which it seems probable that a *quarter of a grain* of strychnia in one drachm of water, at one dose, destroyed the life of a woman, aged 36. The patient was affected with trismus, as the result of a burn, and had been treated with opium. The muscular system was relaxed, and the breathing was short and spasmodic. In ten minutes after the strychnia had been given, the respirations were increased, and almost immediately after the patient was seized with the most violent convulsions, the back being bent forwards (emprosthotonos), the arms raised, and the features contorted. After a time, these spasms ceased for the space of about a minute: they then recurred with extreme violence, and continued until death took place, in about an hour and three quarters. (See 'Med. Times and Gazette,' April 15, 1854, p. 376.) A patient of M. Andral suffered from the most violent tetanic convulsions from so small a dose as *one twelfth of a grain* of strychnia. There are probably some constitutions peculiarly susceptible of the effects of this poison.

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10	Case of Mrs. S. Smyth, 'Pharin. Journal,' 1848, vol. ii, p. 298. An adult female (case quoted at the trial of W. Palmer).	Three grains of <i>strychnia</i> , in liquid, by mistake for salicine. Spasms in from five to ten minutes; screaming; legs drawn up; feet turned inwards; body stiff; easy before death; said "turn me over;" died tranquilly; consciousness retained. Death one hour and a quarter from taking the medicine.	Inspection three days after death. Body healthy; contraction in feet continued, but had gone off somewhat from the rest of the body; no disease in any part; heart contracted, and perfectly empty. The blood was fluid.	No chemical analysis of stomach or contents. (Medicine analysed.)
11	Case in Provincial Hospital, in 1848. 'Med. Times and Gazette,' April 28, 1855. A girl, æt. 12.	One grain of pure crystallized <i>strychnia</i> in pill. Soon afterwards, there was violent sickness; convulsive spasms of a tetanic character followed. She died in one hour and a half.	No report of inspection. Death referred to some undiscovered disease of nervous system — when, four years afterwards, it was found that the bottle labelled " <i>Valerianate of Zinc</i> " from which the pill had been prepared, contained pure <i>strychnia</i> !	No report of analysis.
12	Case reported by Dr. Smith, of Forfar, occurred in May, 1851. 'Edinburgh Monthly Journal,' September, 1851, p. 234. An adult male.	Dose and symptoms unknown. A game-keeper, found dead; a phial labelled <i>strychnia</i> , poison, containing white powder, lying near; deceased had not been seen for four days.  issued on cutting scalp; blood-vessels of brain highly congested; blood dark coloured and fluid throughout the body; lungs healthy, but gorged with blood; heart natural, white sediment. The powder both ventricles much distended; stomach presented a patch of congestion near cardiac extremity, small quantity of whitish powder adhering to membrane; abdominal viscera healthy; spine not examined.  tison, acidulated with sulphuric acid, filtered; treated with milk of lime, concentrated to a thin syrup, and filtered again. The mixture dried at 212°, treated with boiling rectified spirit, and evaporated to dryness. Extract had intense bitter taste of <i>strychnia</i> , and turned bright yellow by active acid. The colour-test did not act characteristically. Dr. Christison remarks, "There is, in fact, a want of positive tests for <i>strychnia</i> when mixed with organic matters, unless the proportion were considerably greater than in this case, so as to admit of some complexity of chemical handling. I think there is satisfactory evidence for a case of suicide or accident. It may be doubted whether this chemical evidence, however, would be enough on a charge of murder, in which there was no proof of the nature of the symptoms."	Body lying on face; hands firmly clenched; feet much extended; head considerably bent backwards; every joint rigid, and some muscles had the condition as if in powerful action dur- boiled with dilute sulphuric ing life; countenance livid, and some- acid; nitric acid added pro- what swollen; dark-coloured fluid blood duced a light orange; tannin bloody points in brain; choroid turgid; caused a deposit of a gray- white sediment. The powder in phial gave similar results. Contents of stomach subse- quently examined by Dr. Chris- tioned again. The mixture dried at	Stomach contained no food. only a few ounces of fluid ex- haling a strong spirituous odour; portion of contents condition as if in powerful action dur- boiled with dilute sulphuric ing life; countenance livid, and some- acid; nitric acid added pro- what swollen; dark-coloured fluid blood duced a light orange; tannin bloody points in brain; choroid turgid; caused a deposit of a gray- white sediment. The powder in phial gave similar results. Contents of stomach subse- quently examined by Dr. Chris- tioned again. The mixture dried at

13

Case reported by Dr. Lonsdale, occurred November 28, 1854. 'Edin. Monthly Journal,' 1855. A strong man, æt 59. One grain and a half of strychnia by mistake for jalapine. Symptoms in death. Brain and medulla oblongata in natural state; heart healthy, empty, one ounce, a thick, grayish-looking fluid, having a slight acid reaction. Stomach and contents boiled in water, acidulated with sulphuric acid; carbonate of lime added to neutralize the acid; evaporated to dryness, and digested with rectified spirit; again filtered, and filtrate evaporated; liquid had consistency of a slight yellowish colour, and a distinct and persistent bitter taste. To the extract, sulphuric acid and bichromate of potash added, gave a purplish tint, speedily changing to a permanent light green. This test, as well as the peroxide of lead and sulphuric acid, failed to give any appreciable change of colour in a small quantity of the extract. Sulphuric acid and bichromate of potash added to an alcoholic solution of strychnia, produced a tinge of purple, rapidly succeeded by the green colour, like the effect on the extract from the stomach.

14

Case reported by Mr. D. G. M'Pherson, occurred at Bristol, in November, 1854. 'Medical Times and Gazette,' December 16, 1854. A male adult. Dose and symptoms unknown. Found dead in bed, body warm; decubitus on his back, which was arched; the occiput was buried in the pillow; heels pressed into the bed; forearms flexed, and fists clenched upon the abdomen; toes drawn to the soles of the feet; eye bright, but not prominent; pupils dilated; features placid; teeth firmly clenched. Inspection eighteen hours after death. Abdomen warm, tense, and hard; body lying on its back, rigid; teeth clenched; eyes a dull, fishy appearance; forearms the flexed, fists clenched; legs forcibly extended; bloody fluid issuing from the mouth. Stomach and duodenum presented red spots of extravasation (congestion?). mucous membrane softened. Both lungs engorged; presenting black patches of pulmonary apoplexy; mucous stomach contained about two ounces of thick fluid, in which were a few grains that, with the sulphuric acid and the bichromate of potash, gave the purple colour of strychnia. The stomach and intestines were of a darkish buff colour. The contents of

No.	Authority and Date of Occurrence.	Dose taken and Symptoms observed. Period of Death.	Post-mortem Appearances.	Analysis and Results.
15	Case reported by Dr. Geoghagan, in 'Dublin Medical Press,' occurred in July, 1855. A male, æt. 26. [A report of this case was not published until the 25th of June, 1856. (See the 'Dublin Medical Press' of that date.)]	<i>Five grains of strychnia.</i> Heard moaning in <i>fifteen minutes</i> ; lying on back in bed; hands clenched; fore-arms flexed; one leg extended, the other in the act of being so convulsively; head drawn back. He died from <i>twenty to twenty-five minutes</i> from taking the poison.	Inspection eighteen hours after death. Body fresh, rigid, resting chiefly on back of head and heels; limbs bent with difficulty; feet extended; face with animal charcoal to recolor; mouth slightly open, with brown matter on tongue, and froth of same colour adhering to the teeth; brain natural, its vessels containing but little blood; lungs moderately congested; the heart was firmly contracted, its cavities contained a very small quantity of dark fluid blood; mucous membrane of stomach softened, presenting dark clay-brown patches; subjacent veins injected; contents partly digested food of a red-brown colour, acid reaction.	Filtered contents of stomach were strongly bitter. Concentrated (acidulated?) agitated with animal charcoal to recolor; afterwards removed from charcoal by boiling rectified spirit; evaporated; dissolved in liquor potassæ, and agitated with ether by Stas's process. The ethereal extract leaves the strychnia sufficiently pure for testing, or the conversion of residue into acetate of strychnia, and a repetition of the process with potash and ether. <i>a.</i> Alkaline reaction and bitter taste of ethereal extract. <i>b.</i> Marked
16	Case of John Parsons Cook; date November 20th, 1855. Æt. 28.	Dose taken unknown. Symptoms on Monday night, the 19th, about an hour and a quarter after some pills had been given by Palmer; violent screaming first heard; beating of bed-clothes with his arms; complained of feeling of suffocation; his head drawn back; moving, as by jumping or jerking, both	First examination six days after death. After death, body rigid, and hands clenched; feet turned inwards; abdominal viscera healthy; stomach containing it. Heated with some ounces of a brown fluid, at dilute sulphuric acid and alcohol, filtered, evaporated; neutralized by carb. potash, and digested in al-	Parts examined for strychnia;—coats of stomach, and two ounces of bloody fluid from the stomach containing it. Heated with dilute sulphuric acid and alcohol, filtered, evaporated; neutralized by carb. potash, and digested in al-



in the head, and all over the body; contained a bilious fluid; kidneys congested; eyes projecting; gested, right larger than the left; rated, and tested by taste, as gasped when he spoke, as if difficult larynx stained with dark blood; lungs well as by sulphuric acid and for him; hands fixed and stiff, asked contained much fluid blood, accounted bichromate of potash, a slight to be rubbed; twitchings in the arms for by gravitation; heart natural size, purple colour produced, but and body during the rubbing; twitched and healthy, its cavities were empty; no satisfactory evidence of all over; quite conscious; all the time blood throughout body fluid, and un-strychnia. Antimony found recognised Palmer, and said "Oh, Pal-coagulated; brain and spinal marrow in these parts. Two drachms mer, I shall die," had a difficulty in healthy; dura mater of brain con- sent), antimony found there- swallowing some pills which were given gested. Second examination two months in. Liver, spleen, kidneys, to him. In taking some liquid, he snapped at the spoon, and seemed to after death. Body and limbs still very and lungs, examined only for bite it very hard, his body was then rigid; hands closed firmly; putrefaction mineral poison. Antimony jerking and jumping; snapped in same not much advanced; spinal cord and alone found in small quantity, way at a glass containing a draught canal examined, both quite healthy. in all the parts submitted to which was given to him by Palmer. examination.

When swallowed, it was vomited immediately, no pills could be found; fit lasted half an hour, then became composed (Mills's evidence.) Twenty-four hours after this attack, from which he perfectly recovered, seized with another and more severe attack, about an hour after taking two pills given to him by Palmer; started up; stiffness of neck, which he asked to have rubbed; swallowed two other pills (ammonia?); uttered loud screams; dreadfully convulsed in all the muscles of the body; opisthotonos; complained of suffocation; asked to be lifted up and turned over; could not be done owing to rigidity of the whole body; quite conscious; heart gradually ceased, and he died tranquilly, about *one hour and a quarter* after taking the pills.

In examining this table of deaths from strychnia, it will be perceived that of the fifteen fatal cases which I have been able to collect by searching authentic records, extending over a period of nineteen years, eleven only, prior to the case of Cook, have occurred in this country. In seven of these cases no analysis was made. In Dr. Blumhardt's case (No 2), although so much as forty grains had been taken, and the patient died in one hour and a half, no strychnia could be found either in the stomach in an unabsorbed, or in the blood in an absorbed state. Although the colour-tests for strychnia were not then known yet the most delicate test for this poison, namely the *bitter taste*, was accessible to the analyst. In fact, according to the recent observation of Dr. Christison, the absence of bitterness is the best negative test of the absence of strychnia. The case is further remarkable from the fact, that although one of the oldest authentic cases on record, there has been none in which the symptoms and appearances have been more accurately or minutely recorded. It appears from this history, that the patient, a young man, must have taken a much larger dose than Cook, and he died from its effects in about the same period of time ; yet, although a special research for the poison was made by a competent person, who had access to the stomach and contents in an unmutilated condition, *not a trace of strychnia was found in the stomach, in its contents, or in the blood*. In case No. 5, except by the bitter taste, there was no evidence of strychnia, and the sulphuric acid is stated to have produced a rose pink colour, without any addition of bichromate of potash. The effect was such as would be produced by salicine and not by strychnia. The tests applied, in the case No. 8, cannot be said to have demonstrated the presence of strychnia in the stomach. In case No. 12, examined by Dr. Christison, the tests applied are tests for brucia, and not for strychnia. Although the man had evidently died from a large dose of strychnia, the colour test did not reveal this, and it is properly stated that such chemical results as these would be of no value, unless there was *proof* from the *nature of the symptoms*. In No. 13, the colour tests were applied to the stomach and contents, and gave some, but not conclusive, evidence of the presence of the strychnia. Strychnia is peculiar in not rapidly reducing chromic acid to the state of green oxide of chrome, and

yet this appears to have been the most striking and prominent colour produced, a result owing to the presence of alcohol or organic matter, and probably the high temperature, caused by the admixture of sulphuric acid. No. 14 is the only case reported to have occurred at Bristol, which I have met with, and it is probably the case referred to by Mr. Herapath, in his evidence. Strychnia was here found in the contents of the stomach by the colour-tests, but the presence of organic matter appears to have affected the results. The case presented no difficulty. A large dose of the poison had been taken—some crystals were left in a drinking glass—the stomach and contents were properly preserved—and there was no poison but strychnia to engage the researches of the analyst. No. 15, the case reported by Dr. Geoghegan, as having occurred in July, 1855, has been published by this gentleman since the case of Cook has drawn general attention to the subject; it is the only one in the table in which strychnia was clearly and unambiguously detected in the stomach, not merely by the colour test, but by those other corroborative characters which render the demonstration of the poison conclusive and satisfactory. It was found in the contents of the stomach and in the coats of that organ; but, as Dr. Geoghegan properly remarks, this must not be regarded as absorbed strychnia, but rather as imbibed from the fluid contents of the stomach. In forming an opinion regarding an analysis, however, it is always necessary to consider the circumstances attending the case. In Dr. Geoghegan's case, the large dose of *five grains* was taken, —there was no vomiting, and the person was dead in *twenty-five* minutes. This gentleman also had, beyond doubt, the contents of the stomach for the purposé of his analysis.

I have records of many other cases of poisoning by strychnia, in which persons have recovered, and one case which has occurred since that of Palmer, in which I am informed that strychnia was detected both in the contents of the stomach and in the blood. The cases of recovery, with one exception, do not throw any light on the question we are now considering, namely, the detection of strychnia in the body. The exceptional case is, however, too remarkable to be altogether passed over. It occurred to a patient in St. Bartholomew's Hospital: the analysis was made by Dr. Stenhouse, Professor of



Chemistry in that Institution, well known for his researches in organic chemistry. A man having taken strychnia medicinally, attempted suicide by swallowing at once *four grains*, mixed with four grains of morphia, dissolved in one ounce of spirit. The usual symptoms appeared in rather more than half an hour. He was brought to the hospital *one hour* after he had swallowed the poison; and by the aid of the stomach-pump, three or four ounces of animal charcoal mixed with water, were injected as an antidote. "The stomach was completely emptied, and during the whole time occupied by this proceeding (twenty minutes), he had no convulsion." The contents thus withdrawn, under the most favorable circumstances, were examined by Dr. Stenhouse, but we are informed "he did not detect any of the strychnia." The patient, it appears, had short paroxysms at intervals after the removal of the contents of the stomach, showing that his system was still under its influence, probably owing to the portion of poison which had been absorbed. The last convulsion occurred three hours after his admission. From that time the recovery of the patient was complete.<sup>1</sup> This case merits a little consideration in reference to the alleged certainty of detecting strychnia in the stomach. It presents us with a simple fact and not a mere speculation. The analysis was performed by a gentleman who has devoted more attention than most chemists in London or the provinces, to organic analysis, and the very means were used,—animal charcoal—for fixing the strychnia, which are recommended by Graham and Hofmann as the most certain and satisfactory. In fact, they were such as many analysts of experience would have employed, supposing the man to have died at the time the stomach-pump was introduced. Nothing is more easy, as experiment will show, than to procure strychnia from animal charcoal thus employed, by the use of rectified spirit; but, although, *four grains* of strychnia had been swallowed, and only an *hour and twenty minutes* had elapsed, not a trace of the poison could be detected in the contents of the stomach. Is it to be supposed that Dr. Stenhouse would have been more successful, in this instance, had he operated on these contents in the dead body in place of the living? I see no reason for coming to this conclusion, and, whatever theory we may advance to account for its

<sup>1</sup> Report of the Abernethian Society, 'Medical Times and Gazette,' April 28, 1855, p. 423.

disappearance, whether by absorption or decomposition, the fact must be taken to prove conclusively, that a large dose of strychnia may in some instances entirely disappear from the stomach in little more than an hour. It may appear surprising that the man should have recovered at all after having taken so large a dose, but it is to be observed that the medicinal dose of strychnia which he had been in the habit of taking for two weeks, had reached half a grain. A report of this case was read before the Abernethian Society, in April, 1855,—a Society which adopted the untisual course of prematurely criticising and condemning on newspaper reports, the evidence of the medical witnesses for the Crown, in the case of Palmer. Their own records would have shown them, that in one material point at least, the medical witnesses were right, and they were wrong; and the verdict of the jury should teach them caution in the future discussion of such matters.

If we refer to toxicological authorities we shall find that there is not one who supports the statement of the chemists for the defence, that, in cases of poisoning, strychnia is not only easily detected in the stomach, but that no conceivable case of poisoning can occur by it in which its presence cannot be determined in the dead body by chemical tests. Dr. Christison, in the latest edition of his work, gives no special process for detecting strychnia, but, under the head of *nux vomica*, observes, "Contrary to what takes place in regard to vegetable poisons generally, *nux vomica* is often found in the stomachs of those poisoned with it."<sup>1</sup> He does not refer to any case in which

<sup>1</sup> 'Treatise on Poisons,' p. 896. The woody fibre of *nux vomica* protects it from the solvent or absorbent action of the stomach, and it thus remains adhering to the mucous membrane, holding a portion of strychnia which a chemist can easily separate and detect, when the strychnia itself, in an independent state, would have been long since absorbed and removed. It is difficult to understand how, in spite of this well-known fact, Mr. Herapath, by his evidence, should have tried to impress the Court with the idea that there is greater difficulty in detecting *nux vomica* than in detecting strychnia. In answer to Lord Campbell, he said, "I can extract the strychnia from the body, *even* where *nux vomica* has been the cause of death." *Ques.* "Do you mean that *nux vomica* is more difficult to discover?" *Ans.* "It is more *complicated* of course." There is no complication in the matter. *Nux vomica* is quite insoluble in all organic liquids, and is not absorbed; the powder, if present, may be easily obtained from the stomach by washing and subsidence. When thus obtained, there is no chemical difficulty in obtaining from a sufficient quantity of it, clear evidence of the presence of strychnia and brucia.

pure strychnia or its salts, taken as poisons, have been found after death in the stomach or tissues.

The counsel for the defence, in referring to Orfila on a point on which Orfila's views were supposed to be favorable to his case, properly described him as having occupied the highest rank among analytical chemists (toxicologists). Orfila, in the latest edition of his '*Traité de Toxicologie*'<sup>1</sup> (1852), revised shortly before his death, gives no case of poisoning in which he has found strychnia in the dead body. Four experiments are related, two performed by himself, and two by others. The experiments consisted in adding small quantities of the sulphate and acetate of strychnia to various mixtures of animal and vegetable matters, allowing them to ferment or decompose during a period varying from a few days to three months, and then applying tests to the filtered decoctions of these mixtures. These tests consisted, not in the use of the "colour-tests," which Orfila does not mention, but in the application of taste to detect bitterness, and in the use of nitric acid to determine the presence of brucia. The results, as far as they went, showed that strychnia is not liable to be entirely removed or destroyed by putrefaction. But in these experiments the tests acted so dubiously, that Orfila felt himself compelled to make the following observations :

"On voit par ces expériences, que s'il est possible de déceler la strychnine ou ses sels au milieu de liquides organiques colorés, il est néanmoins difficile de constater quelquefois, l'ensemble de leurs caractères : on ne saurait donc être assez circonspect lorsqu'il s'agira de se prononcer sur un empoisonnement par cet alcaloïde, et il faudra surtout tenir grand compte du commémoratif et des symptômes éprouvés par le malade.

"Ici comme dans l'empoisonnement par les sels de morphine et de brucine, il ne suffit pas de s'attacher à des phénomènes de coloration : il faut, pour établir l'existence du poison, mettre à nu la strychnine ou ses sels, de manière qu'on puisse constater tous leurs caractères." (Tome ii, pp. 595-6.)

It will be seen, therefore, that there is not a single fact in Orfila's treatise to justify the statement that strychnia is, and

<sup>1</sup> Tome ii, p. 594, Paris, 1852, cinquième édition.



must be, uniformly discovered in every case of poisoning. The adding of strychnia to dead organic liquids and solids, and detecting its presence at different periods subsequently, is one thing,—the searching for it in a dead stomach in which it has been subjected for one or more hours to living processes, is another. Absorption and other vital processes, do not go on in porcelain vessels or earthen jars, as in the living stomach; and the results of such experiments cannot, therefore, throw any light upon the period at which, if introduced during life, we may expect to find the poison in the dead body. Besides, the quantity of salts of strychnia upon which Orfila operated in such a prepared mixture, was not the fifty thousandth, or even the one thousandth of a grain, but thirty centigrammes, or nearly *five grains*! Even when operating on such a quantity of the poison, the reader will mark the caution and circumspection with which this deservedly great toxicologist speaks of the inferences to be drawn from his chemical researches. In the cross-examination of Dr. Rees by Mr. Grove, who has deservedly the reputation of a scientific man and a good chemist, this gentleman, with the work of Orfila before him, put the following questions to the witness:

“Q. You have told us that you consider the poison must be absorbed,—do you know that *when absorbed* it has been found in the blood and tissues? A. I do not know of any satisfactory experiment to that effect. Q. Do you know that Orfila has found it in matter that has been putrefied for a long time? A. I am not aware of it: I do not think of necessity that putrefaction would destroy it: it may. Q. In Mr. Cook’s case putrefaction had hardly set in? A. We had the body approaching the condition, but there was no very marked degree of decomposition.”

The answers given by Dr. Rees were perfectly consistent and proper. But what can be said of a series of questions of this kind emanating from a scientific man? The second question, in reference to the discovery of strychnia by Orfila in putrefied matter, is ingeniously made to follow one in which the counsel had asked the witness whether, when the poison had been *absorbed*, it had been found in the blood and tissues. On the witness replying that he knew of no satisfactory experiment to that effect (a perfectly correct answer, for up to the date of the

poisoning of Cook, strychnia had not been found in the blood and tissues), the counsel immediately suggested Orfila's experiment, the results of which had not the slightest bearing on the detection of absorbed strychnia. Mr. Grove knew at the time, or had the means of knowing, that Orfila's single experiment was performed in May, 1827, *i. e.* twenty-nine years ago, by mixing in a jar about five grains of acetate of strychnia, dissolved in a pint and a half of water, with some intestines (how much is not stated). The contents were exposed for three months, and then examined. In reference to this very experiment, Orfila so entirely distrusted the result which he obtained, that he makes use of the remarkable expressions elsewhere quoted (p. 358), "the phenomena of colour must not be relied on, to demonstrate the existence of the poison:—strychnia, or its salts, must be so reproduced, that *all its characters* may be established."<sup>1</sup> Such are the facts on which Mr. Grove relied to induce the Court and jury to believe that *absorbed* strychnia had been found in the blood and tissues. Assuming that Orfila's experiment, of 1827, proved that unabsorbed strychnia

<sup>1</sup> *Nimium ne crede colori*, appears to be a rule in testing organic alkaloids, on which Orfila very strongly insists. On the value of the colour test for morphia—nitric acid, he observes, "This involves a serious question. Can a medical jurist rely upon *simple changes of colour alone*, as evidence of the presence of morphia, or its salts? Assuredly not, for it is not impossible that, under certain states of disease, the animal fluids may undergo unknown changes, and may give rise to one or more of these reactions. Such a conclusion should never be drawn, until morphia, or its salt, has been separated and obtained." ('Toxicologie,' ii, p. 231.)

In a case mentioned by him, MM. Ruspini and Cogrossi were completely deceived by the colour test for morphia,—iodic acid and starch, on which, on the authority of Liebig and Serullas, much reliance was formerly placed. They found that a decoction of the viscera of a calf, which had taken no poison, gave the same coloured reaction with the test as the supposed poisoned articles which they were engaged in examining. I have since heard of a case in which, from the application of the same test, morphia was supposed to be contained in and eliminated with the urine. It was subsequently found, that both lithic acid and the lithate of ammonia (constituents of healthy urine), produce the same change in iodic acid as morphia, and that this had given rise to the error. These remarks apply to strychnia and other alkaloids, as well as morphia. The colour-tests are useful, when we can obtain an alkaloid crystallized in substance; but the mere indications of colour, although they may give rise to suspicion, cannot be safely relied on as conclusive evidence. M. Devergie remarks ('Méd. Legale,' tom. iii, p. 17), that nothing is so deceitful as an absolute reliance upon colour in testing. Four persons may look at the same coloured product and it will be found to present to each a different shade or tint. This especially applies to those cases in which the quantities are extremely minute.

might be in contact with putrefied animal matter for a period of three months, without being entirely destroyed, it follows that, in order to apply it to the case of Mr. Cook, it must be supposed—1, that deceased had had a dose of *five grains* of strychnia; 2, that from the time of his taking the poison until his death, it had undergone no change in the living stomach; and 3, that the Crown witnesses had in these analyses as certainly to deal with the poisoned contents, as Orfila had with the contents of his jar.<sup>1</sup>

Devergie in the third edition of his work,<sup>2</sup> in his account of strychnia refers to Notus's test, namely, sulphocyanide of potassium; but, although, the "recognised colour tests" had been known for a period of nine years, he, like Orfila, does not even mention them. The whole of his third volume, is devoted to toxicology, and there is no fact recorded to show that strychnia has ever been detected in the stomach, the blood, or other tissues.

M. Galtier, the second edition of whose work was published in 1855,<sup>3</sup> enters minutely, in a volume on 'General Poisoning,' into the processes for detecting the alkaloids, such as strychnia, but he relates no instance in which strychnia has ever been detected in the dead body either in the unabsorbed or in the absorbed state. He condemns, as uncertain and unsatisfactory, any tests applied to liquids or extracts obtained from animal matter, and insists that in organic toxicology it is indispensable to obtain the alkaloids in a pure state, in order that their physical and chemical qualities may be determined.<sup>4</sup>

<sup>1</sup> There is only one experiment in Orfila's work which at all bears on the question. This is in reference to the *sulphate of brucia*. Orfila gave to a dog about five grains of this salt dissolved in two ounces of water. The animal was hanged in two hours. The liver was immediately removed, cut into pieces, and digested in boiling alcohol. After boiling for a quarter of an hour, the decoction was filtered, and evaporated to a soft extract. This was mixed with a little water, and from the action of nitric acid and chloride of tin, it was inferred that it contained brucia. As no attempt was made to get rid of the organic matter, this result cannot be considered satisfactory. Orfila himself objects to any inference in a case of poisoning being drawn from these colour tests so applied. To affirm its presence, brucia must be separated, as such, and its different chemical characters established. ('Toxicologie,' ii, p. 597-9.)

<sup>2</sup> 'Médecine Légale Théorique et Pratique,' par Alph. Devergie, Paris, 1852, troisième édition.

<sup>3</sup> 'Traité de Toxicologie,' 3 tomes, Paris, 1855. <sup>4</sup> Op. cit., tom. ii, p. 250; iii, p. 104.



M. Flandin, another French author, who has written a most elaborate treatise on poisons,<sup>1</sup> enters more into this subject than the preceding authors; but even here there is nothing satisfactory in relation to the question which we are considering. He has "poisoned animals in his laboratory (how many or what animals he does not state) with from three quarters to one grain and a half of strychnia," and he says, "I have constantly found this poison in the contents of the intestines (*matières intestinales*). Will it not equally be found in the liver and other organs into which it shall have been carried by absorption?" He thus answers his own question—"*I have not had time to pursue these researches.*" (Tome ii, p. 255.) Thus instead of a fact we have a speculation. The tests on which he relied are not even stated, but the only one of the colour tests mentioned by him in his list of the chemical reagents for strychnia is sulphuric acid and peroxide of lead. M. Flandin has, therefore, done no more than the witnesses in Palmer's case. He has given comparatively large doses of this poison to small animals, and has then looked for it and found it in the unabsorbed state in the alimentary canal. In his two volumes, he reports no case of poisoning by strychnia in the human subject, in which that poison has ever been found in the dead body.

He quotes the process for the alkaloids recommended by M. Stas, a Belgian chemist, who, in 1847, undertook the chemical investigation relative to the poisoning of the Count Bocarmé by nicotina,—a process which is probably the best that has yet been suggested. "On three occasions," M. Stas observes, "during a period of six years, I have discovered the alkaloids in cases of poisoning: in 1845, at Bruges, morphia was detected in the viscera of a body after an interment of thirteen months; in 1847, morphia was also detected in the viscera of Bureau; and about this period, I detected aconitina in a suspected liquid which had become considerably changed (*profondement altéré*)." The quantity of morphia taken by the deceased persons, the period which they survived, the quantity found in the viscera, the viscera in which it was detected, and the tests upon which M. Stas relied to swear positively to the presence of morphia

<sup>1</sup> '*Traité des Poisons, ou Toxicologie appliquée à la Médecine Légale, à la Physiologie et à la Thérapeutique,*' par Ch. Flandin, tom. i, ii, Paris, 1853.

and aconitina, are not mentioned. Although no cases are given, or facts mentioned, M. Stas sums up his memoir by the general statement—"I have applied the principles just laid down (by his method of research) to morphia, codeia, strychnia, brucia, veratria, emetina, colchicina, aconitina, atropia, and hyoscyamia; and I have been able, without the slightest difficulty, to separate these different alkaloids *when previously mixed with foreign matters.*" These results, however, cannot be taken as referring to the separation of the poisons above-mentioned from the viscera of human beings or animals which had taken them during life; for on this subject there is no account of a single experiment. The analysis refers to the separation "of strychnia and brucia from *nux vomica*, veratria from the extract of veratria, emetina from the extract of *ipecacuanha*, colchicum from the wine of colchicum, aconitina from an aqueous extract of monkshood, hyoscyamia from a very old extract of henbane, and finally atropia from an old tincture of belladonna."<sup>1</sup> Facts of this description have a pharmaceutical interest, but until the results have been verified by repeated trial in the dead body, they are of very little value to the medical jurist. Some of the poisons which he mentions will destroy life in a minute fractional proportion of a grain; and no process, however delicate, can make up for a small quantity of poison distributed by the circulation through an enormous mass of animal matter.

In reference to vague statements of this kind, I agree with M. Galtier, who says, "that authors in affirming that they have *detected* these poisons in the bodies of men and animals, in the matters ejected, or in the contents of the alimentary canal, have in general preserved a discreet silence on their processes for separating them, and on the nature of the tests by which they professed to recognise them."<sup>2</sup>

The most recent practical work on processes for the detection of poisons is that of Dr. Otto, Professor of Chemistry in the University of Brunswick, the discoverer of the colour test for strychnia, (bichromate of potash and sulphuric acid) upon which so much has been lately said and written.<sup>3</sup> This writer,

<sup>1</sup> Flandin, *op. cit.*, tom. ii, p. 143.

<sup>2</sup> Galtier, *op. cit.*, tom. ii, p. 251.

<sup>3</sup> 'Anleitung zur Ausmittlung der Gifte,' von Dr. Fr. Jul. Otto, Braunschweig, 1856.

after speaking of the great difficulties attending the search for strychnia in organic liquids, dwells with minuteness on the method of detecting this alkaloid, and the application of his own colour-test, but he relates no case in which he has ever applied it or known it to be applied, for the detection of strychnia in the absorbed or unabsorbed state in the dead human body.

With these facts before us it becomes necessary to examine the grounds on which it has been alleged that strychnia can *always* be detected in the dead body. The first witness in support of this proposition was Mr. Herapath.

Mr. GROVE.—Q. Have you seen any case in which you have examined the human body where strychnia has been taken? A. Yes, in *one case*. I had the contents of the stomach. Q. Did you recognise it in that state on chemical tests being applied? A. I discovered it in the contents of the stomach. Q. How long after death? A. It must have been three days after death. Q. What was the test you used? A. I used common sulphuric acid and the bichromate of potash (the colour-tests). Q. Have you experimented on animals? A. I have. Q. To a large extent? A. Not to a large extent; sufficient to establish the principle. Q. Can you tell me to what extent? A. In my own experiments and those I have witnessed, eight, nine, or ten. \* \* \* Q. Confine your attention to those which you have analysed? A. There are *two* which I have destroyed myself; they were cats. I gave *one grain* of strychnia in a solid form. In this animal I found in the urine which had been ejected, strychnia; and I also found it in the stomach by the tests mentioned. \* \* \* The second case was also a cat. I gave the same quantity (*one grain*) in a solid form in food. \* \* \* Q. Did you find strychnia? A. Yes; in the urine, in the stomach, and upper intestines, in the liver, and in the blood of the heart. Q. In *all cases* in which you have seen a chemical examination or a search for strychnia, has it been found? Did you detect it by the same tests? A. Yes; but I went a step beyond—"I took extraordinary precautions to get rid of the organic matter." Q. In *all cases which you have seen*, when strychnia has been taken, has the examination been successful? A. Yes; not only strychnia, but nux vomica.



\* \* \* Q. Are you of opinion, as a chemist, that where strychnia has been taken in a sufficient dose to poison (to destroy life?) it *can be detected* and *ought to be detected* by chemical science? A. Yes, up to the time the body is decomposed completely. I mean even where there is putrefaction, &c.

*Cross-examination.*—When you have first experimented for the purpose of finding strychnia, has that been principally in the stomach? A. *Until lately.* Q. *When did you first look for it in the tissues of the body?* A. *On the 8th May.*<sup>1</sup> Q. With a view to this case? A. Certainly; it was with a view of learning everything that I could with respect to strychnia. I have experimented in about nine instances to find strychnia; they were not chemical experiments made by myself but things sent to me to analyse.

MR. GROVE.—Q. What is the smallest quantity you have detected in the stomach? A. I can show you some which I extracted from a dog; *if* the strychnia is perfectly free (from organic matter) I am certain that I could discover the *fifty thousandth part of a grain.* By the ATTORNEY-GENERAL.—Q. Suppose a grain had been administered to an animal, how much should you expect to find in the heart? A. Very little indeed; it is a difficult experiment, and requires great precaution, but it can and has been shown. MR. GROVE.—Q. What proportion? A. I cannot tell; I have no idea, but it must be a very small quantity, supposing that there are twenty-five or twenty-six pounds of blood in the human body. I found strychnia in an ounce and a half of the blood of a dog weighing twenty-five pounds (poisoned by one grain). With the eighth part of the liver of this dog I made four distinct experiments with the four tests,<sup>2</sup> so that I experimented upon

<sup>1</sup> It is worthy of observation, that it was just prior to this date, that the paper elsewhere published (*ante*, p. 327) containing the details of the chemical processes adopted by Dr. Rees and myself, in Cook's case, was sent to the solicitor for the prisoner, for the special information of his chemical witnesses. From that document they would learn, that we had examined the coats of the stomach for strychnia, and *that the tissues of the body* had not been examined for this poison. Their experiments, which must have been somewhat hastily prosecuted, as the trial commenced on the 14th May, may have been for some time contemplated; if so, it is only a very remarkable coincidence.

<sup>2</sup> The four colour-tests, *i. e.*, sulphuric acid with bichromate of potash, peroxide of lead, peroxide of manganese, and ferrocyanide of potassium.

the *thirty-second* part of the liver. I could see the violet colour in that minute portion—it changed to purple, passing to red afterwards.

Mr. RODGERS, the next witness, deposed that he had performed *one* experiment on a dog. He poisoned the animal in December last, with *two grains* of strychnia. He analysed some of the blood when putrid, ten days after the death of the dog (how much blood is not stated). He separated the strychnia, or rather determined its presence, by the colour tests. He *had never analysed any portion of the human subject for strychnia*.

Dr. LETHEBY.—I have witnessed many cases of death by poisoning from strychnia—many of the lower animals—and several cases of poisoning by nux vomica in the human subject, one of which was fatal. \* \* \* I do not hesitate to say, *of all poisons, either mineral or vegetable, strychnia is the most easy of detection after death.*<sup>1</sup> I have detected it in the *stomachs of animals*, in numerous instances. I have detected it in *the blood and in the tissues*. (The date when, the quantity of matter examined, the kind of animal, and the doses given to the animals are not stated.) \* \* \* I have succeeded in detecting very minute portions of strychnia. *When it is pure* it can be detected in very small fractions of a grain, at least the *twenty thousandth part of a grain*; when mixed up with organic matter it would be more difficult of detection, no doubt; still, I can detect the *tenth part of a grain* in a *pint of any liquid* that it may be put into. \* \* \* I have detected it in a pint of the most putrid liquid that I could obtain, in which the tenth part of a grain had been standing for two months.

Mr. KENEALEY.—Q. You have told us you have succeeded in detecting it in an animal which had been killed a month, and was in a state of decomposition; what was the dose you

<sup>1</sup> Dr. Letheby is certainly not of the opinion of Dr. Otto, the discoverer of the very delicate mode of colour testing, on which he (the witness) relies. Writing in 1856, this excellent chemist says, "The separation of very small quantities of the alkaloids (strychnia and morphia) from articles of food, contents of the stomach, &c., requires the greatest experience, and a most practised hand, and yet this separation in the purest condition possible, is necessary, because a very minute intermixture of any foreign substance renders the tests used for their detection, fallacious." ('Anleitung,' p. 88.)

had given? *A.* I gave the animal (a rabbit) half a grain, and *I have the strychnia here within a fraction of what I gave him.* LORD CAMPBELL.—*Q.* In what proportion? *A.* I lost about the tenth part of a grain in the course of the investigation.<sup>1</sup>

The evidence of Dr. Wrightson includes the following points. He stated that he had found strychnia in the blood of a dog poisoned by two grains, in the urine of another dog poisoned by one grain, and in the viscera of a cat poisoned by half a grain. He stated, in answer to a question, “If a man had certainly been poisoned by strychnia he should certainly expect to discover it. *Not generally, but certainly.*”

Mr. Nunneley stated in his evidence that he had experimented with strychnia in sixty animals of various kinds. In the case now known as that of Mrs. Dove, he had been engaged with Mr. Morley, and they had detected, by the colour-tests, strychnia in the contents of the stomach. In this case, however, after the chemical experiments had been performed, four animals were killed with the residue of the contents. He had himself never detected strychnia in the blood or tissues. *He never tried to extract it.* He saw the experiment performed by Mr. Herapath, and the *first time* he had seen it done was *only the day before the trial.*

*Question* by Mr. SERGEANT SHEE.—Supposing death to have been caused by a dose of strychnia-poison, sufficient but not more than sufficient to destroy the animal, in your judgment would it be so decomposed by the process of absorption

<sup>1</sup> It is difficult to make any satisfactory deduction from this evidence. It is not stated how the strychnia was given to the animal, nor from what parts of the body it was subsequently extracted. If strychnia be given wrapped in paper, the greater part may be recovered from the stomach after death (see experiments, *ante*, by Mr. Devonshire and Mr. Horsley, p. 336). If so given, that the greater part is absorbed and removed from the stomach, then it follows that *all* the parts of the body to which the strychnia is carried by the blood, must be made to yield the poison, or so large a proportion could not be recovered. Mr. Herapath found a portion in the urine, and Mr. Rodgers, the preceding witness, states that he has discovered it in the bones. Hence, it follows, that all the excretions, as well as the skeleton must be analysed, before the whole of the strychnia administered can be recovered within a very small fraction of a grain. The result as it stands appears to show that a rabbit may be killed by the tenth of a grain, or if this were merely lost in the investigation, then it follows that the strychnia must have killed the animal by some catalytic property, and after penetrating his whole body, have come out as little changed as silica or spongy platina!



that you would not be able to detect it by these tests (the colour-tests), in any portion of the system? *A.* No. LORD CAMPBELL.—That would be what is called a *minimum dose*, enough and no more.

In answer to a question put by the Attorney-General, this witness stated that the dose which he had generally given in his experiments was “from *half a grain to two grains*.” The animals experimented on were “dogs, cats, rats, mice, guinea-pigs, rabbits, frogs, and toads.”<sup>1</sup>

<sup>1</sup> This witness it will be perceived has had a better opportunity of solving the question now at issue, on the detection of absorbed strychnia, than any one of the chemical witnesses who appeared for the defence. The case of Mrs. Dove occurred in March, 1856. This lady, it is well known, had been poisoned by repeated doses of strychnia, given over a period of a week. She died about twenty minutes after the last dose, which must have been a large one, as the contents of the stomach, after sufficing for numerous experiments and the application of the colour-tests, still contained enough strychnia to poison *four animals*. Mr. Nunneley had taken an interest in Cook's case, and knew that strychnia had not been detected by Dr. Rees and myself. He admitted to the Attorney-General, that he first communicated with the solicitor for the prisoner, Mr. Smith, by sending to him a report of Dove's case. The finding of strychnia in Mrs. Dove's stomach was thenceforth made the great point on which it was alleged that Cook could not have died from this poison. The importance of examining the tissues in the case of Mrs. Dove was therefore obvious, and no case could have been more favorable than this for such a scientific research. Mr. Nunneley had access to the blood, and the whole of the viscera in the case of this lady. He had the most perfect confidence in his processes and tests for this poison, for in one experiment on the stomach of an animal he believed the poison to be present, when Mr. Morley, his coadjutor, doubted the result. He is asked by the Attorney-General: *Q.* Did you know that this poison could be extracted by chemical processes from the tissues? *A.* I thought it *probable*. *Q.* You never tried it? *A.* No. Why, if he thought it “probable,” did he never try the experiment upon the blood and tissues of any one of the sixty animals, poisoned by him with from half a grain to two grains of strychnia, or upon the blood and tissues of the lady, the whole of whose body was placed at his disposal for analysis, at the very time when the country and profession were calling for facts in reference to the detection of strychnia! Instead of resorting to this proceeding, he witnesses an experiment tried by another person only the day before the trial of Palmer, and then, without any direct experience whatever on the subject, swears, in the most positive manner, that no person can die from a minimum dose of strychnia without the poison being detected by those tests (the colour-tests), *in any portion of the system*. This witness, it appeared, voluntarily gave the benefit of his advice and experience to the solicitor for the defence. Supposing the blood and tissues of Mrs. Dove to have been analysed, and strychnia had not been found, or that he and Mr. Morley had differed as in a former experiment about the results, the reader will perceive that all the evidence respecting the extraction of strychnia from the tissues of cats and dogs

It appears to me, it must be obvious from this report and analysis of the evidence of the medical witnesses for the defence, that they have adduced no satisfactory reasons for the strong opinions which they gave on the infallible detection of strychnia in all cases of poisoning by it. The only witnesses who had had an opportunity of making an analysis in the human subject were Mr. Herapath and Mr. Nunneley. In cases presenting no difficulty they found the poison in the contents of the stomach. That strychnia may be detected in this organ in certain cases all the witnesses are agreed; but those for the prosecution think that such circumstances as a minimum fatal dose, absorption during an hour or two that the patient may survive, and the tampering with the stomach and losing its contents, wholly or in part, may lead to an occasional failure of the chemical processes: those for the defence think otherwise. This is the essential point of difference so far as the *stomach* is concerned. Time will probably show which is the more correct view.

As to the *blood and tissues*, not one of the witnesses for the defence had ever made an examination of these parts in the human body for *absorbed strychnia*. Two of them, Mr. Herapath and Mr. Nunneley, had each had an excellent opportunity of determining this question practically in the cases which respectively fell under their notice; but for some reason or other neither took advantage of it.<sup>1</sup> The unabsorbed

hastily made a week before the trial, would have gone for nothing. The opinion which Dr. Rees and I had entertained, in the case of Cook, that strychnia cannot with certainty be found under such circumstances, would have been corroborated, the time of the Court saved, and a great scientific question practically solved.

<sup>1</sup> It is well known that *nux vomica* owes its poisonous properties to the strychnia absorbed from it. Mr. Herapath stated in his evidence, that he had found *nux vomica* in a fox and two dogs poisoned by it. Dr. Letheby had also met one fatal case of poisoning by *nux vomica* in the human subject. How did it happen, that these gentlemen only sought for *nux vomica* in the stomach? *Why not have searched for strychnia in the tissues?* As a matter of science, this was of great importance; but the problem on which so much speculation has arisen was left unsolved when there were favorable opportunities for its solution. I need hardly remark, that the accuracy of these hyper-chemical views would be severely tested by a research of this kind. One hundred grains of *nux vomica* contain only about half a grain of strychnia, mixed with *brucia*, therefore less than half a grain of the poison the properties of which we are now considering. Thirty grains of *nux vomica* (= to one sixth of a grain of strychnia) have proved fatal to an adult. Can

strychnia remaining in the stomach was all that these gentlemen looked for, while at the same time they have both shown an amount of confidence in the processes for detecting it in the parts of the body in which they had never sought for it that is perfectly astonishing. The research for absorbed strychnia in animals was, so far as the evidence goes, not made by any one of these witnesses until after the question had arisen in reference to Mr. Cook's body. The first discoveries of the absorbed poison were made by Mr. Herapath on the 8th of May, the trial on which the evidence was to be given commencing on the 14th! He found strychnia in the blood and liver of one dog, in the urine of one cat, and in the urine, liver, and blood of the heart of another cat. Each of these animals had been poisoned by a dose of *one grain* of strychnia.<sup>1</sup> Mr. Rodgers's experience is confined to the detection of strychnia in the blood of one dog, to which he had given *two grains* of the poison. Dr. Letheby adds to this evidence, that he has detected it in the blood and tissues of animals, the doses given not being stated. It does not appear that he has ever had an opportunity of searching for absorbed strychnia in a human being who had been poisoned by it. Dr. Wrightson detected

the strychnia from a small and fatal dose of *nux vomica* be detected in the blood, liver, and urine? Nothing would be more easy than the performance of Dr. Christison's experiment of injecting the *sixth part* of a grain of strychnia, in solution, into the cellular membrane of a dog. The animal, in his experiment, died in two minutes. There is nothing to prevent the chemist testing the accuracy of his processes, by seeking for absorbed strychnia in such a case in the blood, liver, and tissues generally. Strychnia given in large doses by the stomach, may impregnate the liver and other adjacent organs by exosmosis and thus lead the chemist into the fallacy of supposing that he had discovered "absorbed" strychnia. At any rate, before very strong inferences are drawn from the presence of a minute quantity of strychnia in the "thirty-second part of the liver" of an animal, it is only proper that all possible sources of error should be excluded. Dr. Kidd found that the liver of a dead animal might be impregnated with arsenic by imbibition from the liquids of the stomach. Matteucci long since proved that, if the hind legs of a frog recently killed were immersed for some hours in a solution of ferrieyanide of potassium, every part of the viscera was so penetrated with the salt by imbibition, that by touching them with a solution of a persalt of iron, a blue stain more or less deep was produced.

<sup>1</sup> This, it will be observed, is comparatively a very large dose to give to such animals. It is equal to, at least, five or six grains given to a human being. The Court was desirous of information on the process for detecting strychnia where the dose was at a minimum, and this was the kind of evidence supplied!



it in the blood of one dog poisoned by two grains, and in the urine of another poisoned by one grain; also in the viscera of a cat (absorbed?) destroyed by half a grain. Of Mr. Nunneley's experiments I have already spoken; he has never detected it in the blood or tissues. Even in reference to the bodies of the four animals poisoned by the contents of the stomach of Mrs. Dove, he did not seek for the poison (in the blood or tissues) in any one instance, although this would have thrown great light on the power of chemistry to detect absorbed strychnia in instances in which it was known to have destroyed life.

The whole of the chemical defence resolves itself into this. From experiments made since the death of Cook, on *four dogs* and *two cats* ("a large and tried experience," according to Mr. Sergeant Shee), without a single fact derived from the examination of the human body, the witnesses for the defence considered themselves justified in asserting that no *human being* could die from the effects of a minimum dose of strychnia without the poison being detected.<sup>1</sup> The question then is: Are these facts, even if they were multiplied tenfold, sufficient to warrant the assertion that strychnia, when operating in minimum doses, cannot possibly escape detection in the stomach or in some portion of the system? This question will receive a solution according to the knowledge and experience of those who are engaged in such researches. We have already seen from the record of cases that the poison has not yet been found in the tissues of persons poisoned by it; and further, no toxicologist has recorded an instance in which he or others have succeeded in detecting it.

The case of Dr. Warner (No. 9 in the table, p. 349), who died from the effects of half a grain of sulphate of strychnia, corresponding to 0.43, or forty-three hundredths of a grain of pure strychnia, will enable us to test the accuracy of the opinion thus strongly expressed. This being a soluble form of strychnia, if we assume that the whole was removed from the stomach by absorption in an hour, the proportion of strychnia would not amount to so much as one fiftieth of a grain in a pound of blood, or less than one eight-hundredth of a grain in an

<sup>1</sup> Dr. Letheby did not state in what number of animals he found strychnia in the blood and tissues.

ounce.<sup>1</sup> If, in the short space of twenty minutes, during which the deceased survived, one half only was removed by the absorbents, then one quarter of a grain might be detected in the stomach; but in the blood the proportion would be no more than one hundredth of a grain in a pound, and less than this proportion in any of the tissues. In the experiments on which the chemists for the defence relied in order to support their theory, they gave to the animals, weighing from one seventh to one tenth of an adult human being, doses of strychnia twice and four times as great as the quantity which had actually proved fatal to Dr. Warner!

Dr. Geoghegan, Professor of Medical Jurisprudence at the Royal College of Surgeons in Dublin, observes, in commenting on the medical evidence in this case,—“With the views which would claim for the chemical evidence a positive result in *all* cases of strychnia-poisoning, I can in no wise concur. Nor does the testimony given at the late trial on this head appear by any means sufficient to establish a proposition so striking, and yet so little in accordance with what might, *a priori*, have been expected on physiological grounds. Thus, in the case of the experiments on the part of the defence, the dose administered was always large in proportion to the size of the animal. Hence the only legitimate inference appears to be, that where a similar proportion obtains in the human subject, and under accompanying conditions also alike, the poison should be discovered; and accordingly it was so in the two cases above alluded to, both of them instances of suicide, where usually much more is taken than is sufficient to cause death. In charges of murder by poison, on the contrary, the investigation is not uncommonly conducted under circumstances of a very different kind; *for should the administrator possess a certain degree of skill, the dose may be so apportioned that the detection*

<sup>1</sup> The quantity of blood in the body of an adult is here estimated at twenty-five pounds. Haller considered that it amounted to about one fifth the weight of the body, and thus it would be twenty-eight pounds in a person weighing one hundred and forty pounds. Todd and Bowman give the results of Valentin, who assigns thirty-two pounds for a man between thirty and forty years of age, and twenty-eight pounds for the female; but they say there is no reason to infer that the quantity exceeds thirty pounds. These statements show that the quantity on which the calculation is based in the text, is taken at a low average.

*may become either difficult or impossible.* A case is on record in which *half a grain* of sulphate of strychnia (less than the quantity of the uncombined alkaloid) caused speedy death; and, on the trial of Palmer, another was given in evidence, where the same result ensued from *three quarters of a grain*. Now it must be obvious to any who have practical experience in these matters, that under such circumstances the probability of its detection would be comparatively small, whilst, regard being had to the vastly greater mass of the blood and tissues in which it would in such cases be distributed, as contrasted with those of animals (the dog or cat) made the subject of express experiment, the chance of its unexceptionable discovery in the latter quarters might be equally slight.”<sup>1</sup>

In these remarks we have the opinion of an independent man, of skill and experience, whom those engaged for the defence of Palmer were most desirous of bringing forward as a witness. Feeling, however, from what he knew of the case, that death from the action of strychnia was clearly substantiated, he declined the invitation to attend.

The opinions of the medical witnesses for the prosecution were very much in accordance with the view taken by Dr. Geoghegan. Dr. Christison stated that he should not expect to find strychnia in the body after death where the quantity taken was small. If the excess left in the stomach over that removed by absorption were considerable, he would expect to find it. Mr. Morley said, where small or minimum doses were given, he should sometimes expect to fail in its detection. Hence it follows, if a minimum fatal dose could not always be detected in the stomach, it would be still less likely to be found when diffused through the whole mass of the body by absorption.

But what meaning is attached to the word “detection?” One half of all scientific controversies are properly referred to some misunderstanding of the meaning of the words employed. From the statement made by Mr. Herapath, that he could detect 1-50,000th of a grain of strychnia; and by Dr. Letheby, that he could detect the tenth part of a grain in a pint of any liquid that it may be put into, *i. e.*, when diffused through 87,500 times its weight of organic matter; it is quite clear that the witnesses for the defence rely upon the production of a transient purple colour,

<sup>1</sup> On Poisoning by Strychnia, from ‘Dublin Medical Press,’ 1856, p. 9.



produced by adding to this infinitesimal quantity of strychnia a mixture of sulphuric acid and bichromate of potash, or some chemical agents that will produce a similar colour. There can be no doubt that this is a very "delicate" process : but, in questions of murder, a Court of law requires certainty rather than delicacy. Marsh's process for arsenic will give a metallic deposit when the millionth of a grain is present ; and Reinsch's process will give a coloured deposit on copper when the arsenic is in a proportion almost as small. At first, chemists were inclined to rely upon the visible appearance of such deposits alone, as positive evidence of the presence of arsenic ; and it is well known that such a reliance led even the experienced Orfila into the belief that arsenic was a normal constituent of every human body. This was proved subsequently to be an error by a Commission appointed to examine a scientific question involving such serious consequences. Chemists are now universally agreed not to rely upon the results obtained by these "delicate" processes for arsenic, except when the quantity of deposit is such as to admit of the application of some correcting test or process. Are we to demand less of Otto's colour-test for strychnia than of the processes of Marsh or Reinsch ? In operating on minute quantities of thousandths of grains, it is clear that there can be no corroboration ; the judgment must be based on the change of colour produced : and here wide differences of opinion may arise. A very remarkable example of this occurs in the evidence given by Mr. Morley and Mr. Nunneley, the two gentlemen who conducted the investigation in the Leeds case, and who appeared, the one as a witness for the prosecution, and the other for the defence, at the trial of Palmer. In reference to the detection of strychnia in all cases in which it had been given to animals, the following question is put to Mr. Morley :

MR. GROVE.—Q. I think you said in one or two you speak doubtfully ? A. In one certainly ; in that case we were sure that strychnia had been administered, our doubt was whether it had reached the stomach ; in *that* case I may say there were appearances which a *sanguine eye* might say were those of strychnia—a *more cautious one would doubt*.

The chemical evidence given for the defence would lead to the inference that the colour-test is all in all sufficient

for the detection of strychnia, and that it is exposed to no sort of fallacy. But it is obvious that the relative shades of colour produced will receive different interpretations according to the more or less sanguine views of those who are engaged in the analysis. The question, therefore, for a Court of law is—Is this a satisfactory ground on which to receive a positive opinion of the presence of strychnia on a charge of murder, when the opinion may be the turning point of the prisoner's conviction or acquittal? The practical utility of the colour-test applied to the tissues, and the amount of courage possessed by a witness in relying upon it, cannot be fairly tried until a case of this kind has presented itself.

Certain organic compounds, such as pyroxanthine, salicine, and aniline, produce, in very minute proportions, colours, on contact with sulphuric acid, which might, in some instances, be confounded with the effects of small quantities of strychnia, where an eye was very eagerly engaged in looking for this poison. A drop of cod-liver oil produces with sulphuric acid, with or without bichromate of potash, a play of colours that I have known to be mistaken for the action of strychnia. There is no difficulty in distinguishing any of these substances from each other chemically, but such reactions with organic matter should, it appears to me, teach caution in drawing inferences. Has the liver, tissues, or blood of a person who has been long under a course of cod-liver oil been examined to determine how far a fallacy might arise by over-confidence in the production of colour when sulphuric acid and bichromate of potash are added to the extracts derived from these parts? It is perfectly true that some of the substances mentioned are not likely to be found in the stomach, and that sulphuric acid produces a colouring effect upon them without the addition of bichromate of potash. In reference to the first point, the test has only been known nine or ten years, and it has been so little tried on the fluids of the dead human body, that we can hardly affirm, where the colouring effects are slight, that some combinations of animal matter might not create deception; in reference to the latter point the result will depend on the mode of applying the test. Thus, Schneider, one of the most recent writers on poisons, advises that the supposed strychnia should be first rubbed with peroxide of manganese or other material used,

and sulphuric acid then poured on the mixture.<sup>1</sup> There would be no escape from a fallacy in following such advice; and it is astonishing how easily the eye is deceived when anxiously looking for an anticipated result. The order in which the substances are used in the application of the colour-test is then most important; but the real danger of a fallacy is precisely in those cases in which the quantity of material is so minute that there is not enough for any corroborative test, either by taste or crystallization.<sup>2</sup>

*Experiments on animals.*—At the trial, evidence was given for the prosecution in reference to the detection of strychnia in the bodies of animals poisoned by this substance. It may be proper to place these facts on record, as well as the results of other experiments since made by Dr. Christison and Dr. MacLagan, and by myself, with an anxious desire to arrive at the truth. The animals employed were rabbits, healthy and full grown. The first set of experiments were performed in December, 1855.

EXPERIMENT 1.—*One grain of sulphate of strychnia, mixed with conserve of roses, was given to the rabbit in the form of a pill, which it readily swallowed. The animal appeared very well for seven minutes, when it fell upon its side, and was suddenly seized with a trembling of the whole of the body and limbs, followed by a violent convulsive fit, attended with opis-*

<sup>1</sup> 'Die Gerichtliche Chemie,' Wien, 1852, p. 316.

<sup>2</sup> One of the witnesses for the defence, Mr. Rodgers, professes, since the trial, that he has extracted strychnia from the *bones* of animals poisoned by it, and that in cases of suspicion there may be an expectation of finding it in any undecayed parts of skeletons. Is it really strychnia that has been thus extracted, or is it the *ignis fatuus* of the colour-tests which is misleading this experimentalist? The careful experiments of Millon and Laveran show that in mineral poisoning it requires a period of *several months* for the metal (antimony) to *enter the bones*. Mr. Rodgers is dealing with a poison which kills by minutes, and yet the osseous structure is supposed within this brief period to become so impregnated with the poison, as to yield strychnia on chemical analysis! An experienced chemist informs me that he has tried Mr. Rodgers's process by hydrochloric acid, and it did not succeed. Dr. Stevenson Macadam found that this process caused a failure in his experiments. At present the statement appears to me, so far as the colour-tests are concerned, to be a *reductio ad absurdum*. I shall be induced to modify or withdraw this opinion, when this gentleman has poisoned an animal with the strychnia extracted from the bones of a poisoned animal.



thotonos. The fore and hind legs were rigidly extended, and the body hard and stiff. When forcibly straightened, the head was again drawn back, and the spine assumed the form of a bow. There were repeated jerking motions of the legs, followed by short intervals of relaxation. The spasms were brought on by the slightest causes. Just preceding a convulsive fit, the animal uttered a cry, which was several times repeated; this was indicative of pain. The eyes were wide open and protruded, the pupils dilated; the breathing was difficult, and entirely abdominal. After a succession of fits, at shorter and shorter intervals, the animal died in a violent convulsion, in which the body was drawn into a bow, and the limbs were perfectly fixed. Its death took place, *i. e.*, its heart ceased to beat, six minutes after the commencement of the spasms, and *thirteen* minutes after swallowing the poison. When examined, after a few minutes, the body was found perfectly rigid, in the attitude in which the animal died. This rigidity lasted several days. The cavities of the heart were distended with blood partially coagulated. The lungs were congested, the liver natural. The stomach and intestines presented an opaque-white appearance. The blood-vessels were distended with dark-coloured liquid blood. There was no remarkable fulness of the vessels of the brain or its membranes. The upper part of the spinal marrow and its investing membranes were quite healthy.

The stomach was secured by ligatures, and, with its contents, removed for analysis, as well as the liver, lungs, and heart, with the blood contained in the last-mentioned organ.

EXPERIMENT 2.—A pill containing *one grain* of Morson's crystallized strychnia was given to this animal. Although the strychnia appeared to be of good quality and was found to contain but very little brucia, no symptoms were observed for the long period of an hour and five minutes. A pill containing a grain of another sample of strychnia (making *two grains* in the whole) was then given to it. In *nine minutes* after this second dose the animal was suddenly seized with a convulsive fit, by which its body was jerked violently off the table on which it had been placed. There was the same general tremor over the body and limbs as in the preceding case, appearing like a rip-

ple passing over the surface of water. There was opisthotonos; the fore legs were quite rigid and stretched in a straight line, a little inclined backwards, with the most extreme tension; the intercostal muscles were fixed, so that the breathing was entirely abdominal. The eyes were fixed and prominent, a condition especially observed at the time of the attack of convulsions. There was at this time a frequent cry, as from great pain. When the body was straightened, the tail and head were speedily drawn back, and the spine arched, in the most complete state of opisthotonos. The heart pulsated rapidly; during the fits its beats were 140 in a minute, and in the slight intervals of relaxation, 108. At times its action was hardly perceptible; but on the recurrence of a fit, its beats were so frequent that it was difficult to count them. There was a flickering motion of the muscles of the lower jaw and face. After a succession of fits, the heart suddenly ceased beating, and the animal died, thirteen minutes from the time of the commencement of the symptoms, and *twenty-two minutes* after the second dose of poison had been given to it. The body of the animal became rapidly rigid in the position in which it died; and in twenty-five minutes, while still warm, it was so rigid that it could be held out by the head and chest, or by the haunches, in a straight line, without collapsing. It retained this rigid condition for several days.

The heart was full of blood on both sides. The right lung was emphysematous (distended with air), the left lung natural. The liver was healthy. The blood was dark coloured and partially in a coagulated state. The stomach was very pale on the exterior. The blood-vessels were distended with dark-coloured blood. The intestines were healthy; the spleen was small and collapsed. The brain and spinal marrow were healthy; the membranes were somewhat congested.

The parts removed for analysis were the same as in the preceding case. In addition, a small quantity of blood was collected from the heart and vena cava for special examination.

EXPERIMENT 3.—*One grain* of strychnia was given to this animal in a pill. The symptoms appeared suddenly in *nine* minutes; they ran through their course in eight minutes, and the heart of the animal had entirely ceased to beat in *seventeen*

minutes. There was the same order as well as intensity of symptoms as in the preceding cases—opisthotonos, occasional cries of pain, and violent tremors of all the voluntary muscles of body and limbs, just preceding the convulsive fit. The pulsations of the heart during the fit reached 160. The body became stiff and rigid while warm, and remained as stiff as a board during many days. The jaws were fixed. Before death there were twitching motions about the muscles of the face.

The appearances in the chest and abdomen were similar to those met with in the other cases. The brain and spinal marrow were natural. There was no congestion of the membranes.

EXPERIMENT 4.—This animal took, in the form of a pill, *half a grain* of pure sulphate of strychnia. It had been twenty-four hours without food. The symptoms were similar to those already described; they appeared suddenly in *ten* minutes; in eight minutes from their commencement, and in *eighteen minutes* from the time of taking the dose, the animal was quite dead. The appearances in the chest and abdomen were the same as in Experiment 3. The brain and spinal marrow were in a healthy condition.

EXPERIMENT 5.—*Half a grain* of strychnia was given. The animal moved about, without appearing to suffer, for ten minutes. At this time it appeared to have an unsteady motion on its legs. Every attempt to move was attended with slight involuntary movements of the fore and hind legs. When moved, these irregular motions were well marked, and the animal appeared to cling by its feet with an unsteady hold to the table. At *twelve minutes* after taking the poison it was seized with a convulsive fit, and fell on its side in a state of opisthotonos. There was an interval of relaxation; but slight motions made near the animal, or touching it very gently, brought on the spasms with violence. The whole of its skin appeared highly sensitive. In a few minutes, and after several struggles with its hind legs, which did not appear to be under the control of the will, the animal was enabled to raise itself and walk about. It appeared as if it would recover. By



touching and moving it, other convulsive fits supervened; and in one of the most violent of these it died, eleven minutes after the commencement of the symptoms, and *twenty-three* minutes after the administration of the poison. Particular attention was given to the state of this rabbit at the time of death, and it was observed to be quite flaccid in its body and limbs. This condition lasted but for a short time. The animal was placed on its back, and the legs raised for the purpose of making an examination of the chest. In about ten minutes it became, while still warm, perfectly rigid in this raised attitude, the fore legs remaining stretched upwards, and wide apart. The rigidity in this animal had considerably decreased in two days, and had nearly altogether disappeared in four days.

The heart and lungs were full of blood. The stomach presented the remarkable paleness observed in the other cases. The brain and spinal marrow were healthy.

*Analysis.*—In Experiments 1, 2, 3, the stomachs were found full of green food in a partially digested state. In Experiment 4 there was but a small quantity of food. The stomach and contents in each case were digested in distilled water, acidulated with a sufficient quantity of diluted sulphuric acid to dissolve strychnia at a moderate temperature. The decoction was filtered, and the residue washed with warm distilled water until all soluble matters were removed. By evaporation, a clear yellow-coloured acid decoction was obtained, amounting to about six drachms. The liquid of No. 1 had a decidedly bitter taste; this was well marked in No. 2; but in Nos. 3 and 4 it was not perceptible to Dr. Rees or myself, although a friend imagined that he perceived it in No. 3. Carbonate of potash gave no precipitate in the decoctions, even after they had been allowed to stand for many hours. The extracts obtained from the evaporation of these decoctions were digested in alcohol, and the bitterness was perceived in the spirituous, as in the watery, solutions of Nos. 1 and 2 only. The process already described (*ante*, p. 327) was then pursued, as in the examination of the stomach of Cook. Faint, whitish, uncrystalline deposits were obtained by evaporation on glass, which, when treated with sulphuric acid and bichromate of

potash, gave the following results: In Nos. 1, 3, and 4, no change of colour upon which any reliance could be placed,—the bichromate was slowly converted to green oxide of chrome. In No. 2 the broken crystal of bichromate soon became surrounded with a rich violet-coloured liquid, passing rapidly through a purple to a light red colour. In the whole of these cases the hearts, lungs, and livers of the animals were separately examined for strychnia, and in No. 2 the blood collected from the heart and vena cava was also examined. The aqueous and alcoholic extracts had no bitter taste, and yielded no indication of strychnia by the application of the colour-test.

The process pursued in the analysis of the stomach was, for obvious reasons, the same as that adopted in Cook's case, but in reference to the *tissues*, we also employed animal charcoal for the purpose of separating the strychnia, according to the plan of Graham and Hofmann. The total absence of bitterness in any one of the extracts of the blood and tissues showed that there could be no appreciable quantity of strychnia. This, according to Dr. Christison's recent trials, is perceptible in cases in which the colour-test fails.<sup>1</sup> It is a property which cannot be concealed by organic matter, and to remove every objection on this ground, the one hundredth of a grain of strychnia was used as a counter-test. When mixed with the residue of each dial glass, in which the colour-test had failed to act, the colours were immediately produced in a clear and decided manner.

Although the tissues of No. 1 gave no indication of strychnia, the acid aqueous extract of the liver, heart, and lungs acquired by concentration, and without the addition of bichromate of potash, a rose-pink colour. It resembled the appearance presented by solutions of strychnia or veratria in sulphuric acid after exposure to heat, but there was no perceptible bitterness. It did not exist in the cases of the other rabbits, although treated in a precisely similar manner, and it may suggest a reflection as to the occasional production of colour in an extract from a dead body from the use of chemical reagents alone.

No analysis was made of the rabbit in Experiment 5. The

<sup>1</sup> He has found no indication by the colour-test beyond the 15,000th grain of pure strychnia in *distilled* water, while he perceived the sense of bitterness in *distilled* water when the strychnia formed only the 40,000th of a grain. Taste is therefore in his opinion the best "guide test."

body of this animal was opened, and the viscera, after examination, were left exposed to undergo putrefaction. There was no perceptible difference observed in the time of commencement, or in the progress of putrefaction. It was noticed, however, that hundreds of larvæ of the *musca carnaria* and other insects were living and thriving in the midst of all the viscera of this animal. Strychnia, if present, certainly did not affect them.

Subsequently to the trial I performed an experiment with a still smaller dose of strychnia than any which had been given in the preceding experiments, or by the chemists for the defence.

EXPERIMENT 6.—On the 3d of June, 1856, I gave to a full-grown healthy rabbit, which had been recently fed, *one quarter of a grain* of pure *sulphate of strychnia* dissolved in a few drops of distilled water. The animal swallowed the whole, excepting a small portion which adhered to the glass. It remained active for about *fifteen minutes*; at that time it appeared to be more easily startled by slight noises; its movements were tremulous, and it seemed to be unsteady in moving its hind legs. Soon afterwards it trembled violently, or started when touched. Slight twitchings appeared in the limbs at intervals on its attempting to move, or on making a noise. Eighteen minutes had now elapsed, and it had not been seized with a well-marked convulsion of the trunk. It was gently lifted from the table by the ears to be placed on the floor, when it was suddenly seized with a most violent paroxysm of convulsions. The hind and fore legs were rigidly stretched out, and there was complete opisthotonos. Its eyes protruded, its breathing was difficult; the pulsations of the heart could not be counted; the head and tail were drawn backwards as if by a tightened bowstring, with occasional slight intervals of relaxation, and in this state it died, two minutes after the commencement of the convulsions, and *twenty minutes* after taking the poison. Immediately after death, it was noticed that the whole body was flaccid, and there was a general relaxation of all the voluntary muscles; but the body speedily stiffened, and, what appeared remarkable, the fore limbs altered their position and became rigidly stretched out. In eight minutes from the time of death, while the body was still



warm, this rigidity of the muscles was perceived over the greater part of the trunk. On the 5th of June, the back and hind and fore legs were still rigid. On the 7th, the weather having been in the mean time very warm, the rigidity had in great part ceased; it was then only perceptible in the joints of the hind and fore legs.

At this date the body was examined. The stomach was pale, but much distended with food (probably amounting to half a pound of hay and green food). The coats were softened at the greater end. The intestines contained chiefly air; the lungs were collapsed, and of a bright red colour; the heart on both sides contained blood, partly coagulated; the blood in all other parts liquid and dark coloured. The parts of this animal taken for separate analysis were—1. The stomach and contents. 2. The blood of both cavæ, as well as that obtained from the right cavities of the heart, amounting with the coagula to two teaspoonfuls. 3. The lungs, the heart emptied of blood, the liver, and two kidneys; these organs being placed together for one experiment. 4. The whole of the muscles of the hinder extremities. The process pursued was that recommended by M. Stas.<sup>1</sup> Instead of using tartaric acid, however, I employed acetic acid, having found, by preliminary experiment, that this acid acted well on small quantities of strychnia. The concentrated acetic liquid obtained from the aqueous and alcoholic extracts of the different parts examined was rendered alkaline by potash, and then shaken with ether; the ethereal liquid was poured off, evaporated, and the residue tested. 1. From the stomach and contents whitish deposits of a semi-crystalline character were obtained. Sulphuric acid and bichromate of potash, as well as peroxide of manganese and the ferricyanide of potassium, gave, with these deposits, the violet, purple, and red colours indicative of strychnia. The alcoholic and aqueous extracts had a bitter taste. The quantity of strychnia obtained from the bulky contents of the stomach was estimated at not more than one hundredth part of a grain. 2. The blood, carefully examined by the same process, gave not the slightest indication of strychnia, either

<sup>1</sup> This process, first suggested by the Belgian chemist for alkaloidal poisons generally, will be found described in Flandin's '*Traité des Poisons*,' tome ii, p. 137; also '*Edinburgh Monthly Journal*,' 1852, vol. xv, p. 313.

by bitterness in the liquids, or by the action of sulphuric acid and bichromate of potash on the residue obtained by ether. 3. The lungs, heart, liver, and kidneys. These parts, similarly treated, gave alcoholic and aqueous extracts having no bitter taste. The residue left by ether was in the form of circular white specks, which, when examined by a quarter-inch power, had no distinct crystalline form, but were obviously granular. When tested with sulphuric acid and bichromate, neither the blue nor the purple colour appeared, but after a time a reddish-brown streak formed around the fragment of bichromate of potash. This was examined by a friend, and we came to the conclusion that the result was by far too doubtful to infer the presence of strychnia. That any organic matter could not have obscured the colour was proved by the counter-test of adding to the acid a minute portion of strychnia. 4. The muscles gave aqueous and alcoholic extracts which had no bitterness whatever, and the ethereal residues gave no effects with the tests indicative of strychnia.

Without any knowledge of my experiment, Dr. Christison, in company with Dr. Maclagan, had performed one of a similar kind, the results of which he communicated to me on the 13th of June.

EXPERIMENT 7.—A *quarter of a grain* of pure strychnia, obtained from Morson, made into a pill with bread-crum, was thrust down the gullet of a rabbit. There was decidedly no effect for twenty-five minutes. In ten or fifteen minutes more, no one but a familiarised observer would have noticed anything wrong; but when the animal was raised or pushed, its limbs were tremulous and stiff. In one hour the first spasm occurred; in three quarters of an hour more it was dead, *making one hour and three quarters* from the time of taking the poison until death. This, as Dr. Christison remarks, coincided as nearly as possible with the time of commencement and the time of termination of the symptoms in the case of John Parsons Cook. The body remained quite flaccid for five or six minutes, and then passed through the usual stage and usual degree of rigidity for two days. The heart, examined twenty-four hours after death, was pretty full of *fluid* blood.

The stomach contained a large quantity of food. This, with the contents, was examined for strychnia by Stas's process, the whole of the steps being followed as described by him. Strychnia was found unequivocally by taste and the bichromate test, in the contents of the stomach.

Not a trace of strychnia could be found by the application of these tests to the liver, treated scrupulously by the same process. Not a trace could be found by the bichromate test in the muscles, of which a great quantity were used. The residuum of the process was too scanty to allow of our trying the taste-test in the residue from the muscles.

Dr. Christison observes in reference to this experiment, which was carefully made, not to support a theory but with a purely scientific object, "I can have no doubt that death may be occasioned by strychnia, in circumstances admitting of ample time for full absorption, and that in the tissues it may be impossible to find strychnia by Stas's method."

These two experiments, with nearly the same dose of strychnia upon the same kind of animal, were conducted independently, and it will be observed that the chemical results were as nearly as possible the same. The sulphate of strychnia employed by me, would correspond to about four tenths of a grain of pure strychnia; therefore, the dose was smaller than in Dr. Christison's case; it was given in perfect solution, while Dr. Christison gave the strychnia in a solid form. The smaller quantity, and the fluid form, would be more favorable to removal by absorption; not to mention that the few drops of solution in my experiment, had to traverse the whole of the gullet of the animal, while the pill would probably reach the stomach in the form in which it was administered. These differences will account for the more rapid supervention of symptoms and death in my experiment, as well as for the smaller quantity of strychnia discovered in the contents of the stomach; but it is perfectly clear from the results, that in a small dose, whether given in perfect solution and destroying life rapidly, or in a solid form and destroying life slowly, an animal may die from strychnia, *and no trace of the poison be found in the tissues.* It may be found in the stomach in larger or smaller quantity, according to whether it be given in solution or as a solid, and whether we have or have not the stomach and its contents in an un-



damaged state. In my experiment, the animal died in twenty minutes, and certainly not more than the 100th of a grain of strychnia was separated from the contents of the stomach. Who can doubt, as so large a proportion of the poison had disappeared in this short period, that if the animal had survived an hour, none would have been found in the stomach? Again, if out of 39-100ths of a grain which had thus been lost from the stomach, in twenty minutes not a trace could be found in the heart, lungs, liver, kidneys and blood, is it probable that after sixty minutes, the removal of another 100th of a grain from the stomach would have made any practical difference in the negative results of the analysis? None would have been found in the tissues and none in the stomach.

In Mr. Herapath's experiments, the smallest dose given was four times as great; and in Dr. Wrightson's single experiment on a dog, eight times as great as in these experiments performed by Dr. Christison and myself, and yet these witnesses asserted that, when given in a *minimum* dose to kill, strychnia must be detected in an absorbed state in the tissues! When this was stated upon oath, to influence the minds of the jury to adopt the theory set up for the defence "no strychnia found,—no poisoning," the witnesses had *not even tried a minimum dose* on animals, and therefore were certainly not in a position to swear to the results of an analysis.

I subjoin a tabular statement of these experiments:—

*Before the Trial.*

No.	Dose.	Commencement of Symptoms.	Total period until death.	Analysis.	
				Stomach and Contents.	Blood and Tissues.
No. 1	1 gr. Sulphate Strychnia, solid.	7 minutes.	13 minutes	Detected by taste only.	Nil.
" 2	2 gr. Strychnia, solid.	9 "	22 "	By taste and Bichromate.	"
" 3	1 gr. Strychnia, solid.	9 "	17 "	Nil.	"
" 4	$\frac{1}{2}$ gr. Sulphate Strychnia, solid.	10 "	18 "	Nil.	"
" 5	$\frac{1}{2}$ gr. Strychnia.	12 "	23 "	No analysis.	No analysis.

*After the Trial.*

" 6	$\frac{1}{4}$ gr. Sulphate Strychnia in solution.	15 "	20 minutes	By taste and Bichromate.	Nil.
" 7 (Dr. Christison.)	$\frac{1}{4}$ gr. Strychnia, solid.	60 "	105 "	"	"

Facts cannot neutralize each other; and if in adapting their experiments to meet the real question at issue, the chemists employed for the defence could show that under a minimum dose of the poison they can detect strychnia in the tissues of an animal killed by it, still this would not affect the results obtained independently by Dr. Christison, Dr. Maclagan and myself. It is difficult to conceive any number of affirmative instances which would justify them in laying down as an immutable chemical law that in poisoning by strychnia the poison must always be detected. The assumption of superior skill or superior means of research could not be met by any argument; and, in reference to the search for strychnia, can hardly be claimed by gentlemen who have had so little experience of the various processes for detecting it in the tissues, that one had not sought for it until six days before the trial of Palmer; and the other had not tried the experiment when he had had ample opportunity, but had seen it performed by another just twenty-four hours before the trial commenced!

Since the occurrence of Cook's case, I have received various communications from scientific men in reference to the detection of strychnia in the blood and tissues. Dr. Ogston, of Aberdeen, informs me, in the case of a man, who died in about twenty minutes after taking a quantity of strychnia (the dose not accurately known) that, besides detecting it in the stomach, he found traces of the poison in about four ounces of blood.

Dr. Macadam has also found strychnia in the blood and tissues of animals, in several instances, both with large and small doses. In one instance, it was detected in the tissues of a cat, to which a quarter of a grain, mixed with a grain of muriate of morphia, had been given,—the symptoms came on in forty-three minutes, and the animal died in fifty-six minutes. In another instance, half a grain was given to a dog,—the urine voided only *nine minutes* afterwards, was found to contain strychnia, a fact showing how rapidly the poison is diffused and passes out of the body. It is remarkable, too, that up to this time, the dog had manifested no symptoms of poisoning by strychnia. In seventeen minutes, another half grain was given; and, in another minute, tetanic spasms with the usual symptoms came on. The animal was dead in forty minutes from the time of taking the first dose. From the results ob-

tained in another experiment, he has drawn the conclusion that strychnia, taken in small doses at intervals, is absorbed and retained in the system, notwithstanding that it is thus so rapidly eliminated by the urinary secretion. He gave to a horse thirty-two grains of strychnia in divided doses, the animal having in the first instance taken twelve grains. An hour elapsed before the first symptoms were observed; and the animal died tetanic in two hours. He found strychnia in the contents of the stomach, the muscles, the blood, and the urine contained in the bladder, but he did not detect it in the liver, lungs, spleen, kidneys, or heart.

Dr. Cowan, of Glasgow, has furnished me with the results of some experiments lately performed by himself and Dr. Lawrie. Three dogs were poisoned with a quarter of a grain of strychnia; two of these were examined by Dr. Anderson, and one by Dr. Penny. There was unequivocal evidence of the presence of strychnia in the stomachs of the three dogs. Dr. Anderson found traces of it in the liver. Dr. Easton found it in the urine of one of the dogs, while under the influence of chloroform. Dr. Penny examined with great care the brain and spinal marrow of a dog poisoned with strychnia, but failed to discover any trace of the poison.<sup>1</sup>

Mr. Horsley, of Cheltenham, has communicated to me the results of his observations, which were the subject of a paper read at the late meeting of the British Association (August, 1856). In three experiments on cats, to which he had given doses of strychnia, varying from one quarter of a grain to one grain, the animals having died after some hours, he could not detect any trace of strychnia in the bodies. In one experiment he gave *two grains* of strychnia in a pill with conserve of roses, wrapped in blotting paper. *Five hours* elapsed without any symptom of poisoning showing itself; in the morning the dog was found dead. On inspection, the right side of the heart contained no blood (the auricle and ventricle were empty) while the left side contained blood in a partly coagulated state. When the stomach was opened, the pill was found still enclosed in the paper wrapper. Mr. Horsley subsequently recovered from it one grain and a quarter of strychnia, thus showing

<sup>1</sup> Cases of Poisoning by Strychnia, with Experiments, &c. 'Glasgow Medical Journal, part xiv, July, 1856.



that three quarters of a grain had been removed by solution and absorption, over a period of upwards of five hours, and had poisoned the dog. The contents of the stomach gave slight indications of strychnia; but no portion of absorbed strychnia could be detected in the blood of the animal, or in the tissues of any part of its body. They were examined both by chloroform and ether; but the results were equally negative. Mr. Horsley sent to me a portion of the blood removed from the body of this dog. In the dried state it weighed 168 grains, corresponding to about two ounces. This was examined by Stas's process with potash and ether, but there was not the slightest indication of the presence of strychnia, either by the taste-test or by sulphuric acid in conjunction with bichromate of potash, peroxide of manganese, or ferricyanide of potassium. This, then, confirmed the result previously obtained by Mr. Horsley. From other experiments which he has performed, Mr. Horsley thinks that strychnia may, in certain cases during absorption, enter into intimate combination with the albuminous parts of the tissues, and thus be removed from the sphere of ordinary chemical analysis. This, however, involves another branch of inquiry. The fact derivable from these researches is, that he failed to find strychnia, in cases in which he knew that it had caused death, and in which he was diligently looking for it.

The conclusions to which it appears to me this inquiry leads, are—

1. That strychnia may be found in the stomach, as in other cases of poisoning, when it has not been entirely absorbed, and the stomach and contents have been properly preserved for analysis.

2. That in some cases, when given in small doses, and in other cases even in large doses, although it may be detected in the stomach, it cannot be detected in the absorbed state in the blood and tissues.

3. That there are no facts derived from experiments on animals or from observations in the human subject, to justify the statement that in *all* cases of poisoning by strychnia, the poison must, by proper chemical processes, be certainly detected.

4. That in strychnia-poisoning, as in morphia- and other forms of poisoning, a person may live a sufficient time for the poison to be entirely removed from the stomach, and in this case he may die without a trace of strychnia being found in the blood, tissues, or any part of the body.

*What becomes of the Strychnia in a case of poisoning in which it cannot be detected by chemical analysis?*

This question, as it will be perceived, was much dwelt upon by the counsel for the defence. Under chemical advice, he undertook to give a plain and decided answer to the effect that strychnia was in its nature utterly indestructible, that it could not be lost when once in the body, by digestion, absorption, diffusion, decomposition, elimination in the living, or by putrefaction in the dead body. In fact, when once in the body, and it had worked as an instrument of death, it was just as indestructible as rock crystal or the diamond, and that the unerring tests of chemistry might in all cases bring it to light. It is only fair to state, that he had not a single physiologist to advise him with respect to the correctness of this opinion, and, for the purpose of the defence, he was bound to believe the statement of his chemical advisers.

The defence was laboured to a most extraordinary extent on this curious physiological question, simply in order to prove that Cook could not by any possibility have died from strychnia. Strychnia, the jury were told, although an organic compound of four elements, carbon, oxygen, hydrogen, and nitrogen, was quite indestructible under any circumstances. It had been sought for by gentlemen who were described as well skilled in analysis, but not a particle could be extracted from Cook's body. If he died of it, it must have been there: and if there, it must have been found.

I have elsewhere remarked, that one strange fallacy pervaded the whole of this reasoning. The only part of Cook's body examined for strychnia, for reasons already assigned, was the *substance of the stomach*, and yet the argument was so put, that the jury were led to believe that every portion of the body had been searched for strychnia, and none was found! In being required to explain, why, when a *minimum* dose of the

poison proved fatal, it might not be found in the body, as in a case like that of Dr. Warner, I stated that the strychnia might be partially changed, and thus withdrawn from the ordinary processes of analysis.

Q. In addition to this distribution of the half grain over the whole system (*i. e.*, in the small proportion of one fiftieth of a grain to a pound of blood), in your opinion, does that undergo decomposition, as it mixes itself with the animal tissues? A. I believe it *partially* undergoes *some change* in the blood. Q. Would that increase the difficulty of detecting it in the tissues? A. It does. I have never heard of its being separated in a crystallized state from the tissues.

These answers were immediately seized by the counsel for the defence, and applied to Cook's case; but in a most untruthful manner. I had not said that strychnia was *entirely destroyed*, but that it was *partially changed*; statements leading to wholly different conclusions, because, assuming that strychnia has been procured from the tissues and bones in the ready manner in which recent experimentalists affirm, my statement is perfectly compatible with the detection of an *unchanged portion*, while the theory of entire destruction, invented by the learned sergeant, *pro hac vice*, would be quite inconsistent with its discovery in the blood or tissues. To Cook's case, for the reasons stated, this question of partial change could have no application whatever.

I may here dismiss, briefly, another question, which occupied much of the time of the Court, in the examination of witnesses, to no purpose, so far as the case of Cook was concerned,—namely, the supposed loss of strychnia by *putrefaction*. A large mass of scientific evidence was brought forward to show that in solids or liquids highly putrefied, strychnia, if once placed there, might still be found and its properties demonstrated by the colour-tests. The body of Cook had not undergone any internal change from putrefaction. He died on the 20th of November (a cold season of the year), and the viscera were under examination on the 28th. There was not the least ground for supposing that putrefaction had interfered in any way with the detection of an organic or an inorganic poison. Dr. Rees and myself, had given no opinion on the effects of putrefying matter on strychnia, and the proved detection of it in dead dogs and in decomposed blood after long periods, was



quite unnecessary. I have myself detected strychnia in the stomach of a dog when in a highly putrefied state, and although I cannot go to the length of the witnesses for the defence, in asserting that this alkaloid altogether resists putrefying changes in the midst of dead animal matter, I believe that it is not easily destroyed by the process. Their experiments do not, however, bear out their conclusions. Not one of them has shown, that after such exposure of strychnia, the *whole remains unchanged*. Their facts, as far as they go, are quite consistent with partial destruction even by the putrefactive process.

On the other question, of partial change in the living body, a few words may be said. We have no fact to show that strychnia undergoes *digestion* in the stomach. The gastric juice is a powerful agent; but, so far as I know, its action on strychnia has never been determined by experiment. The acids of the stomach dissolve strychnia, and place it in a state favorable for removal by absorption.

The rapidity with which strychnia is absorbed and diffused through the body, must vary according to many circumstances. An experiment of Dr. Macadam's, already referred to, shows that the poison not only may pass into the blood, but that it passes out again by the kidneys in the very short period of *nine minutes*. This may happen, provided the poison comes in contact with the surface of the stomach, and there is no mechanical or other cause to interfere with its absorption. On the fact of its diffusion, there is one set of experiments by Mr. Blake: he found, on introducing the nitrate of strychnia into a vein, that the action of the poison on the spinal cord was manifested by tetanic convulsions in sixteen seconds in the horse, in twelve seconds in the dog, in six and a half seconds in the fowl, and in four and a half seconds in the rabbit. Severe symptoms cannot be produced until the poison is diffused through the circulation; and the more rapidly it enters the blood, the more speedily do the effects appear. This shows how largely absorption must be concerned in the operation of the poison. Dr. Christison killed a dog in *two minutes*, with the sixth part of a grain dissolved in alcohol, injected into the chest, and a wild boar was killed in *ten minutes* with one third of a grain. An instance has been privately communicated to me, in which a man died in *ten*

*minutes* from a dose of ten grains! This is the most rapid case of death yet known; and there must have been here very speedy absorption and diffusion. Dr. Harley injected one twelfth of a grain of acetate of strychnia in solution, into the jugular vein of a full-grown dog; in *four seconds* the animal became tetanic, and in twenty-eight minutes it died.

In reference to the rapid *elimination* of the poison, we have the evidence of those gentlemen who state that they have found it in the urine. In Dr. Macadam's experiment it was found, as above stated, in the urine of a dog to which half a grain of strychnia had been given on liver, only nine minutes before. The remarkable feature in this experiment is, that no symptoms of strychnia-poisoning had up to that time manifested themselves in the dog.<sup>1</sup> Hence, assuming that there was no fallacy, it follows that the poison begins to be thrown off by the blood before this fluid has acquired a sufficient quantity by absorption and accumulation, to produce symptoms.

This statement is important in reference to poisoning by strychnia, since the collection and examination of the urine while the person is living, might, according to the results of this experimentalist, furnish evidence of the poison being in the system. It also opens to view another question in reference to a supposed *partial change* in the poison, during its distribution through the circulating system. This is what may be called decomposition. Dr. Macadam's experiment clearly shows that strychnia, as such, may be in the blood in sufficient quantity to be thrown off by the urine, and to be detected in that fluid, and yet no symptoms of strychnia-poisoning will exist. To what can this unexpected result lead? It appears to me to be one of two conclusions: 1st, either that the blood is not sufficiently saturated with strychnia to produce the usual effects on the spinal marrow; or, 2dly, that in poisoning by strychnia, a certain time elapses before the blood undergoes such a change by the presence of the poison, as to cause the symptoms peculiar to strychnia-poisoning.

It will be desirable, before speculating on this point, to refer to a few facts regarding the detection of strychnia. In the experiments performed by Dr. Christison and myself, in which a quarter of a grain of strychnia was given to rabbits,

<sup>1</sup> 'Pharmaceutical Journal,' August, 1856, p. 124.

a small quantity, the surplus of that which had gone into the blood, was detected in the stomach, but not a trace in the blood or tissues. In Dr. Harley's experiments, just now related, a dog was killed by the twelfth of a grain of acetate of strychnia, injected into the jugular vein. This gentleman states that he lost no time in making a minute examination of the blood. He employed Dr. Marshall Hall's physiological test for this purpose, but although this is an exceedingly sensitive test, there was no effect; thus showing that there was no strychnia in the blood. The poison, therefore, in these cases, must have been either decomposed, or so diffused that the quantity was too small to admit of detection. Other facts of a similar kind might be quoted. The result of the above experiments is, that when introduced directly into the blood in *minimum* fatal doses, strychnia cannot be found in that fluid, even when death has occurred in twenty-eight minutes. When introduced into the stomach in *minimum* fatal doses, after variable periods, there may be sufficient left in that organ to enable the experimentalist to say that strychnia is present, but not a trace can be found in the blood or any parts of the body. Again, when strychnia has been given medicinally, it cannot always be found in the urine.

What becomes of the strychnia? Is it that there is a limit to the action of the tests, beyond which they cease to reveal its presence, although it is there; or is it to be admitted that it has undergone a change? The theory put forward in the defence, that strychnia is as unchangeable in the animal body as rock-crystal, would, if it were well founded, forbid our entertaining the first supposition. Tests which act up to the 50,000th of a grain upon a substance that is perfectly indistructible, and even resists the action of oil of vitriol, can never be supposed to fail! The only conclusion is, then, that the poison must have undergone some change, owing to which it can no longer be reproduced as strychnia.

There are some facts in support of this view, derived from the recent experiments of Drs. Macadam and Harley. Dr. Harley found that the flesh of animals killed by *minimum* doses of strychnia, did not act as a poison to other animals. He fed a hedgehog on poisoned flesh for a period of fourteen days, without being able to detect the slightest symptom of poisoning.



The poison must, therefore, he concludes, have been either decomposed, or it was not present in sufficient quantity.<sup>1</sup>

I have already had occasion to refer to Dr. Macadam's experiment. He killed a horse with thirty-two grains of strychnia, which can hardly be called a minimum dose. He fed a large-sized terrier dog for two weeks on the flesh of this horse; the animal eating every day during this period two pounds of muscle. The terrier dog, he says, lived and thrived on the flesh, and did not betray the faintest shadow of tetanic symptoms. He states that, on analysis, he found distinct evidence of strychnia in the muscle and blood of the horse.

These results appear to me to favour the view that strychnia, when absorbed and diffused through the circulating system, undergoes some change, by which, although part of it may still exist as strychnia, a portion is so altered or decomposed, that it no longer possesses a poisonous action on animals. Either this view must be adopted, or we must be prepared to say that strychnia, unabsorbed or as it is lying in the stomach, is a poison; but when absorbed and deposited in the tissues, although still existing entirely as strychnia, it ceases to exert a poisonous action.

Does strychnia produce any change in the blood? By some, the dark colour and fluidity occasionally observed in this liquid, are ascribed to the direct effect of this and other alkaloidal poisons. I have frequently examined the blood of animals poisoned by strychnia by the microscope, but could perceive no change in the globules. In a fatal case of poisoning by this substance which occurred to Dr. Ogston, he states that the blood-globules under the microscope appeared swollen, and their outline irregular; but this, he thinks, may have been due to spontaneous changes after removal from the body.<sup>2</sup> In Dr. Blumhardt's case (No. 2 in the table p. 346), it was observed that the blood drawn while the patient was living and labouring under the effects of the poison, presented some peculiar characters. It came from the vein dark-coloured, and of tarry consistency; and there were contained in it a number of minute bladders

<sup>1</sup> 'Physiological Action of Strychnia,' p. 15. This interesting monograph was published in the 'Lancet' of the 7th and 14th June, 1856.

<sup>2</sup> 'Lancet,' April 19, 1856, p. 428.

of gaseous matter, appearing to indicate that it had undergone chemical change.<sup>1</sup>

That strychnia acts as a poison, by producing some change in the properties of the blood, either vital or chemical, is, I believe, a generally received view among physiologists. There is no other conceivable theory which will explain its physiological action as a poison on the nervous system. To suppose that it alters or affects the blood without in itself undergoing some change, is contrary to the generally admitted doctrines of chemistry. It is also contrary to our knowledge of the chemical properties of strychnia and the alkaloids. The extraordinary changes of colour which strychnia undergoes by contact with sulphuric acid and the peroxides in the application of the colour-test, are properly ascribed to its combination with nascent oxygen. In taking oxygen from these substances, is it to be supposed that it undergoes no change in itself? Is it in accordance with the laws of chemistry, that A can affect B, without B affecting A? Are the coloured compounds of strychnia and oxygen still in the form of strychnia the poison, or have they undergone a change? The amount of this conversion in the application of the colour-test, is well known to be dependent on the relative proportions of strychnia and nascent oxygen produced from the substances employed.<sup>2</sup> An ignorant or careless manipulator might, it is true, on finding strychnia to be present, affirm that the alkaloid had undergone no change; when, in fact, part of it only was there, and part of it had become converted into other compounds, no longer possessing the properties of strychnia in its pure state. The application of this reasoning is obvious. There is in the blood nascent oxygen or oxygen in a state of high chemical tension for combination with the carbon and hydrogen of the waste tissues of the body. Strychnia, as it passes into the blood by absorption, must have its molecules split infinitesimally. These molecules there meet with oxygen. Considering the remarkable affinity which strychnia shows for

<sup>1</sup> 'Wibmer Arzneimittellehre und Gifte,' art. "Strychnos," p. 258.

<sup>2</sup> Strychnia readily removes the colour of permanganate of potash, in the cold, by abstracting oxygen. The sulphate of strychnia discharges the colour almost instantaneously. On evaporating the solution, and on filtering it to separate peroxide of manganese, a whitish residue is subsequently obtained, which gives the coloured reactions of strychnia; but, so far as I could judge, diminished in effect, probably by reason of the loss of the portion which had become oxidized.

oxygen out of the body, would it be a matter of great surprise that it should combine with, and remove from the blood a portion of that element on which all vital actions and the proper secretion of nervous force depends? If the oxygen of the blood can combine with the carbon and hydrogen of the tissues, there is nothing to prevent its combination with the carbon and hydrogen of part of the strychnia. It is not necessary that the whole of the absorbed strychnia should be thus changed. The conversion of a part would satisfy the theory, and the altered blood would thus become the poison affecting the motor tract of the medulla oblongata, producing an increased secretion of the nervous force which excites the voluntary muscular system.

But there is another mode in which the blood may be affected. According to Mialhe, strychnia is one of those poisons which forms an insoluble compound with the alkalies of the blood, and it thus affects the organic constitution of this important fluid.<sup>1</sup> Mr. Horsley considers, from his experiments, that strychnia enters into intimate combination with albumen, and that it is thereby so changed, in certain cases, as not to be discoverable in the tissues when it has caused death. I have elsewhere alluded to these experiments (*ante*, page 389). Supposing these views to be confirmed by further observation, it is probable that strychnia, besides its assumed effects on the oxygen of the blood and its action on the alkalies in that liquid, may further influence the condition or proportion of the serum.

Liebig, writing in 1842, says, "With respect to the action of other nitrogenized vegetable principles, such as quinine, the *alkaloids* of opium, &c., which manifests itself not in the process of secretion, but in phenomena of another kind, physiologists and pathologists entertain no doubt that it is exerted chiefly on the brain and nerves. This action is commonly said to be dynamic, that is, it accelerates or retards, or *alters* in some way the *phenomena of motion in animal life*. If we reflect that this action is exerted by substances which are material, tangible and ponderable, *that they disappear in the organism*, that a double dose acts more powerfully than a single one, that after a time a fresh dose must be given if we wish to produce the action a second time; all these considerations, viewed chemically,

<sup>1</sup> 'Chimie appliquée à la Physiologie et à la Thérapeutique,' par le Dr. Mialhe, p. 524, Paris, 1856.



permit only one form of explanation—the supposition, namely, that these compounds, by means of their elements, take a share in the formation of new, or in the transformation of existing brain and nervous matter.”<sup>1</sup>

Liebig, therefore, evidently considers that the alkaloids, under which head strychnia and brucia would be included when absorbed, undergo changes of conversion, and that their poisonous action on the body is really due to the changes thus produced.

As far back as 1824, many experiments on the loss of the alkaloids by absorption and by changes produced in the blood were made by Lassaigue, Dublanc, and other French chemists, the question having excited some interest in consequence of the trial and execution of Dr. Castaign. M. Dublanc gives the following as the results of his various experiments: “When the acetate of morphia has been absorbed, it will no longer be found or produced in a crystalline form from any of the liquids or tissues of the animal body. It appears to me to be demonstrated that, during absorption, this alkaloidal salt is rendered so diffusible that the aggregation of its constituent molecules is destroyed, and I much doubt whether it will be possible to find reagents capable of restoring cohesion to these divided molecules, so as to cause them to reassume the crystalline form. I am led to believe that when crystallized morphia is produced from the liquids of the stomach it is only a portion of that quantity which has *not been absorbed*. With this exception it will be found only dissolved, and appreciable by no test except by the blood-red colour given by nitric acid.”<sup>2</sup>

Although this theory of the partial loss of alkaloids, by absorption and diffusion in the blood, has been before the scientific world for the long period of thirty-two years, and has been widely promulgated by Liebig and his disciples within the last fourteen years, it was described by Sergeant Shee and his chemical witnesses as something quite new, which they had all heard of for the first time at the trial of Palmer! Had any one of his professional advisers been known as a physiologist, or as ever having given attention to physiological chemistry, this statement might have excited surprise. As it is, it shows

<sup>1</sup> ‘Animal Chemistry or Organic Chemistry, in its applications to Physiology and Pathology,’ by Justus Liebig, p. 182, London, 1842.

<sup>2</sup> ‘Considerations Medico-Chimiques sur l’Acetate de Morphine,’ par le Dr. Vassal, p. 97, Paris, 1824.

that theories propounded by well-known chemists of repute may be entirely unknown to those who are put forward as "adepts" in chemistry for the instruction of a Court of law. It is not expected that gentlemen of the legal profession should be acquainted with these scientific theories, but it is expected that they should be better advised regarding them than they appear to have been on this occasion.

There are few physiologists who doubt that all poisons act through the blood, that they alter its physical or its chemical properties, manifested by fluidity and change of colour; and that the poisonous substance itself simultaneously undergoes a change. This has been established by experiment in reference to oxalic acid, alcohol, and sulphuretted hydrogen; and, according to the statements of Mialhe, the compounds of arsenic and phosphorus with hydrogen affect the vital properties of blood by the removal of oxygen. As an additional proof, M. Bernard has recently found that the cyanide of mercury is resolved into hydrocyanic acid in the capillary system of the lungs. There is, therefore, reason, from analogy, to believe that strychnia may undergo some change in this fluid. The fact that it can be demonstrated to exist in the blood and tissues proves nothing to the contrary, because this may be a portion of the unchanged or undecomposed poison. The question can only be determined by experiment; and it remains to be seen whether, under a *bonâ fide* minimum dose, the whole of the strychnia which has destroyed the life of an animal can be recovered from the dead body in its original state. This is by no means probable, if we may judge from careful experiments already made with minimum doses of the poison. It certainly was not proved, or rendered even probable, by the evidence given for the defence at the late trial.

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There was practically no defence,—no answer to the charge of murder brought against William Palmer by such an overwhelming mass of evidence as was produced at this trial. The "incredibly rash" opinion which the learned counsel charged me with having given "upon the loose evidence of chamber-maids, and waitresses, and housekeepers," against the opinion of the medical man, Mr. Bamford, who attended Cook in his

last illness, and with no encouragement from Mr. Jones,—was immediately adopted from an examination of the depositions and without any communication with me, by Sir Benjamin Brodie, Dr. Christison, Dr. Todd, Dr. Rees, Dr. Daniell, Mr. Curling, Mr. Solly and others. In dealing with the medical evidence for the prosecution, as I have already shown in several instances, the learned counsel for the defence had an unfortunate failing of misstating and misrepresenting facts of the greatest importance in the case; but he had quite persuaded himself that there was no crime,—that Cook had died a natural death, and that his client William Palmer was perfectly innocent. Thus, in addressing the jury, he said, “I commence his defence, I say it in all sincerity, *with an entire conviction of his innocence*. I believe there never was a *truer word* pronounced than the words which he pronounced, when he (the prisoner) said *Not Guilty* to this charge. Further, I will give you this proof of the sincerity with which I declare upon this evidence *my conviction of his innocence*, that I will meet the case of the prosecution *foot to foot at every stage*.” How was this promise maintained? Either by a complete evasion of every material circumstance adverse to his case, or by a misstatement of those medical facts on which the guilt or innocence of the prisoner really turned. No reason was given for the purchase and stealthy procurement of strychnia at such an unseasonable time. Not one of the acts of the prisoner in reference to his interference with the dead body, the analysis, or the correspondence relating to it, received any reasonable explanation. The attempted bribery of the post-boy, with a view to the breaking of the jar containing the viscera, was converted into a mere wish to break the neck or legs of a troublesome old stepfather who was so suspicious, and, as it has now been proved, justly suspicious, of the conduct of his client the prisoner!

If we turn to the theory of the cause of death, irrespective of poison, we find a most inadequate suggestion,—excitement arising from the deceased winning a race, manifesting itself by violent tetanic convulsions a week after the occurrence, on two separate occasions, with complete recovery in the interval; and this, too, occurring in a young and comparatively healthy man, who had never been subject to excitement or had had any kind of fit previously! Some of the learned counsel’s witnesses



admitted that the symptoms were consistent with death from strychnia: one, who denied this, said they were consistent with nothing that he ever knew: others, again, assigned widely different causes incompatible with each other. The argument based on the non-discovery of poison in the stomach completely failed; it was not even supported by the evidence of his own witnesses; and an examination of their evidence shows that they had no facts in reference to *minimum* doses on animals, and none whatever in reference to the human subject,—on which to base their opinions.

The last argument in favour of the innocence of his client was certainly of a most extraordinary kind. A letter was read which the prisoner had written to his intended wife about six years previously, and before his marriage,—as a proof of his ardent affection at *that* time; and that he was not, therefore, likely to commit such a crime as that imputed to him by this charge six years subsequently! It was well known, however, to the whole world, that a bill for the murder of that wife by poison had been returned against him; and that, in the event of an acquittal on the charge of murdering Cook, he would have been immediately tried for this crime.

It is unnecessary to carry this examination of the case further. It is, however, a subject of regret that in defending prisoners, some learned counsel are unable to perceive that they should not convert themselves into witnesses and asseverate on their own belief, the innocence of a person whom they are simply retained to defend. Their duty is clearly to see a man *tried according to law and not condemned contrary to law*. A species of wild license, however, is sometimes indulged in by certain members of the bar, where a defence is desperate, which cannot fail in the end to be attended with evil results to society. The denunciation of medical witnesses as conspirators to destroy the life of an innocent man without motive;—the coarse imputations of rashness, ignorance and prejudice, on men who simply perform a duty—always painful, sometimes dangerous in the nature of the investigations required, but absolutely necessary to the safety of society—reflect disgrace on the mode in which we profess to inquire into truth. Learned counsel, who are members of an honorable profession, should bear in mind that the medical profession is equally honorable; and that its members

have a claim to be treated with proper respect. A case of poisoning by strychnia may possibly occur hereafter under circumstances more nearly touching the private feelings of the counsel who defended Palmer in this case, than the death of the unfortunate John Parsons Cook. A medical man may be called in; Is it to be supposed that he will easily forget the charges of "incredible rashness" and "unheard-of indiscretion," brought against witnesses who, in Palmer's case, only performed a public duty,—even although the verdict of a jury and the voice of the country subsequently confirmed the correctness of their views? Will the learned counsel then be inclined to receive an opinion based, as it may be, upon the statements of chambermaids and waitresses, instead of a committee of surgeons or physicians who cannot always be present to witness an act of murder,—or will he again reject it as rash and indiscreet? The course pursued on this occasion by the learned sergeant, will, I fear, have the evil effect of stopping the expression of such a free and independent opinion as every medical man so situated not only ought to give but to be protected in giving. It will cause him to look more closely to consequences as affecting himself and his future position. The mode of dealing with the witnesses for the Crown, in Palmer's case, will show him that in fearlessly performing his duty to the country he cannot rely on receiving protection from violent, unjust, and unmerited attacks.

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#### APPENDIX.

MR. POLAND has kindly communicated to me the following valuable statistical results, obtained from an examination of 68 cases of tetanus recorded at Guy's Hospital since the year 1825.

63 traumatic—57 died; 6 recovered.

3 questionable as to exposure or injury—2 died; 1 recovered.

2 idiopathic—1 died; 1 recovered.

*Inspections.*—Of these, only 27 were inspected, and the heart noticed in 14; of which, 13 were traumatic, and 1 idiopathic. All died during the paroxysm; there was no case of examination where death took place from exhaustion.

*The mode of dying* of the 60 cases, as far as can be relied upon by the eye-witnesses were—

8 from exhaustion.  
24 during the paroxysm;  
28 not noticed.

*State of the heart in the 14 cases—*

- 1, nothing remarkable.
- 1, stated to be large.
- 1, right side flaccid, and left firmly contracted.
- 1, right side slightly loaded.
- 3, right side full of coagulum.
- 6, both sides contained blood; one of these cases *idiopathic*:  
  - In 1, the blood was dark and fluid, and full in quantity.
  - In 1, right side more than usually distended with dark fluid blood; on the left side the same, but smaller in quantity.
  - In 1, the right side had a firm coagulum, and the left some soft clots.
  - In 1, firm clots on both sides.
- 1, heart violently contracted (laryngotomy performed in this case).

At page 312, some remarks were made on the fulness or emptiness of the heart as an indication, or the contrary, of strychnia-poisoning. In death from strychnia, the cavities of this organ have been sometimes found empty, and at other times full; and the facts collected by Mr. Poland from the records of Guy's Hospital show that there is no uniform or constant condition of this organ in fatal cases of ordinary tetanus. Generally speaking, more or less blood has been found in the heart; and in a recent case communicated to me from another source, blood was found in this organ. It is worthy of remark, that in the single fatal case of idiopathic tetanus, blood was contained on both sides of the heart; and in one instance only the heart was found strongly contracted. The empty condition of the heart in the case of Cook was, therefore, more adverse to the theory of death from tetanus, whether of idiopathic origin or as a result of ulcers, than it was to the theory of death from strychnia. In fact, the tabulated cases show that in strychnia-poisoning, emptiness of the heart is an occasional appearance, while in tetanus as a disease it is very rare. It is highly probable that the condition of this organ varies with the mode of death, whether the tetanus arise from disease or poison.

Since these remarks were written, I have had an opportunity of reading an account of the experiments of Dr. Pavy, published at p. 411 of this number of the Reports. The results show that strychnia has no direct action on the heart, and that in animals the arrest of the functions of this organ is the indirect effect of the suspension of respiration by spasm of the respiratory muscles. The occasional emptiness of the heart may, Dr. Pavy thinks, be accounted for by some slight contractions of this organ taking place subsequently to the death-spasm, as a result of a few automatic efforts at respiration. It is not so much with the cause, as with the fact that a medical witness has to deal. The cases of strychnia-poisoning tabulated at p. 346, show clearly, whatever may be the cause, that the absence of blood from the cavities of the heart is certainly not inconsistent with death from strychnia, as the witnesses for the defence wished to make it appear. Whether asphyxia is or is not in all cases the immediate cause of death in human beings poisoned by strychnia,



is a question purely physiological. It had no practical bearing on the case of Cook, and I have not, therefore, considered it necessary to make it a prominent subject for discussion in this paper.

The 27 inspections furnish us with additional facts of interest in reference to certain questions which have been examined in this paper.

*Duration of the cases.*—The periods at which the disease proved fatal varied from four or five hours in one doubtful case, to twenty-eight days, this being the longest period from the commencement of the attack in which the disease proved fatal. The single idiopathic case terminated fatally in ten days. The periods were as follows; 1 proved fatal in 40 hours; 1 in 50 hours; 2 in 2 days; 2 in 3 days; 5 in 4 days; 2 in 5 days; 2 in 6 days; 4 in 7 days; 2 in 10 days; 1 in 12 days; 1 in 14 days; 1 in 21 days; 1 in 22 days; and 1 in 28 days.

I subjoin a brief history of the case which proved fatal in four or five hours. It can hardly be denominated a case of tetanus.

“Female, æt. 9. Burn over upper part of body and both arms. Progressing favorably until ninth day of residence in the hospital, when the limbs became suddenly and rapidly rigid, and the deglutition exceedingly difficult. *There were no convulsions.* Death in four or five hours. The brain and cord were found healthy, but firm. The heart had firm clots on both sides.”

*Condition of the Brain and Spinal Cord.*—Among the 27 inspections there were 14 in which the brain and spinal cord were *healthy*. Of the remaining 13 cases, the cord was found softened in 2; one of these having proved fatal in ten days, and the other in four days. In 4 cases the cord was congested, these having proved fatal in fifty hours, in two days, in three days, and in seven days respectively. In 2 cases the cord is described as darker than natural; one having proved fatal in fourteen and the other in two days. In the latter case the cord was firm. In 4 the cord was found pinkish, and either natural or firmer than natural; and in 1 case, which proved fatal in four days, the examination having been made forty-one hours after death (in August, 1855), the cord was decomposed, while the body was still rigid.

No granules or deposits were found in any one case. The reader will perceive from this statement of facts observed in the human subject, that there is no uniform appearance in the brain or spinal marrow attendant on death from tetanus. In the majority of cases these parts were healthy, the inspections having been recent. Let these facts be contrasted with the suggestion made in the defence of Palmer to the effect that had a recent inspection of the spinal marrow been made in the case of Cook, a (natural) cause of death might have been revealed!

# ANALYSIS OF THE WATER

OF THE

## GREAT GEYSER, ICELAND.

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By ALFRED S. TAYLOR, M.D., F.R.S.

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A SAMPLE of water taken from the Great Geyser Spring, in Iceland, was sent me by Dr. Little, of H.M.S. Coquette. It was collected on the 16th June, 1856, the temperature of the water being 190 degrees, and the temperature of the air forty-seven degrees. The quantity sent to me amounted to eight fluid ounces.

The water was bright and colourless; and on standing, it deposited no mechanical impurity. It had no odour; but a somewhat saline and alkaline taste. Its specific gravity, the thermometer being 73° (August, 1856), was a little above that of distilled water.

Its gaseous contents were oxygen and nitrogen; in the small quantity examined, there was no trace of free carbonic acid. It did not become turbid on boiling. It was alkaline, and retained an equal degree of alkalinity both before and after boiling.

Its hardness, as determined by the soap-test, was half a degree. It was quite as soft as distilled water; but the softness was owing to the absence of the salts of lime and magnesia, and to the presence of carbonate of soda.

On evaporation, it left a dry, almost white crystalline-looking residue, having a slightly brownish tint from the presence of a trace of oxide of iron. This residue was entirely mineral. There was no indication of the presence of organic matter either animal or vegetable. The residue was not deliquescent. Calculated for the imperial gallon it weighed 106·6 grains.

A chemical examination of the saline residue obtained from this water, showed that the only alkaline base contained in it

was soda. This was associated with carbonic acid, chlorine, sulphuric, and silicic acids; and the mineral constituents of the water, besides a minute trace of oxide of iron, were chloride of sodium, carbonate of soda, sulphate of soda, and silica,—the last being the preponderating mineral ingredient. The quantity of saline residue was too small to allow of a strictly accurate determination of the proportions of these ingredients, excepting the silica; but of the 106·6 grains, there were—

	Grains.
Soluble in Water . . . . .	58·6
Insoluble in Water and Acids . . . . .	48·0
Grains . . . . .	<hr/> 106·6

The concentrated aqueous solution had a very strong alkaline reaction, owing to the presence of carbonate of soda. The sulphate of soda was estimated to form one fourth of the soluble salts, and the chloride of sodium one third; hence the constitution of the water based on these estimates would be:—

	Grains.
Carbonate of Soda . . . . .	19·53
Chloride of Sodium . . . . .	24·42
Sulphate of Soda . . . . .	14·65
Silica and Matter insoluble in Water and Acids . . . . .	48·00
Oxide of Iron (traces) . . . . .	0·00
Grains in Imperial gallon . . . . .	<hr/> 106·60

Many years ago an analysis of one of the Geyser waters was made by Dr. Black, and he found its constitution to be as follows, the quantities being reduced to the Imperial gallon:

	Grains.
Carbonate of Soda . . . . .	6·51
Muriate of Soda . . . . .	17·22
Sulphate of Soda . . . . .	10·22
Silica . . . . .	38·22
Alumina . . . . .	3·36
Grains in Imperial gallon . . . . .	<hr/> 75·53

The quantities of saline matter contained in the Imperial gallon differ, a result which is to be expected in waters taken from the same or similar springs, at periods very remote from



each other ; but the ingredients found, are, for the most part, the same ; and there is this striking feature in both analyses, that the water holds in a dissolved state a very large proportion of silica. The largest amount of silica found in cold spring water is about  $\frac{1}{10,000}$ th part, or seven grains in a gallon ; in ordinary spring or river water, the proportion rarely exceeds two grains in the gallon.

Although silica is not very soluble in water, nearly 8000 parts of water being required to dissolve one part, even when the silica is in a state most favorable for solution, there are conditions connected with the water of the Geysers, which render this substance very soluble. These are the large amount of carbonate of soda present, and the high temperature of the water in the earth. As to the first condition, it has been recently determined that the solvent action of water on silica is great in proportion to the amount of carbonate of soda or alkaline carbonate present. The silica appears to be dissolved as silicic acid, at any rate it does not separate by evaporation as silicate of soda ; and it does not displace the carbonic acid from the carbonate when evaporated to dryness, and the residue is moderately heated. Another condition which affects the solubility of this substance is the high temperature of the water. In July, 1846, Bunsen found the temperature of the water (before an eruption) at the bottom of the Geyser, (about seventy feet) to be 261 degrees. At this temperature, the pressure is equivalent to two atmospheres, or thirty pounds on the inch ; and this pressure, combined with the heat, is probably favorable to the solution of silica in water.

The source of this silica appears from recent researches to be—the volcanic silicious minerals known as phonolite, basanite, and dolerite, which are contained in the upper layers of the volcanic soil around the springs. Analysis has shown that unaltered phonolite contains 72·3 per cent. of silica, while the rock altered by exposure to the water, contains only 65·8 per cent.

The silica contained in the Geyser water is not precipitated by mere cooling, but it is slowly deposited on all surrounding objects, as the water evaporates, and the solvent,—carbonate of soda is withdrawn.

REMARKS  
ON THE  
PHYSIOLOGICAL EFFECTS OF STRYCHNIA,  
AND THE  
WOORALI POISON.

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BY F. W. PAVY, M.D. LONDON:

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THE *strychnos nux vomica*, and the *strychnos toxifera*, belonging respectively to East India and British Guiana, and associated together in the same botanical order, yield materials producing exactly opposite effects upon the animal frame. Both are of the most poisonous and virulent character, and act upon the spinal portion of the cerebro-spinal system; but, whilst the one excites, and leads to muscular contraction, the other depresses, and leads to muscular paralysis. *Under the influence of both, the heart remains perfectly free.* Indeed, it is more especially from the discrepancy of opinion that was recently noticed, with regard to the condition of the heart under the influence of strychnia, that the results of my experiments have been shaped into the present communication.

STRYCHNIA.

If strychnia be introduced into the system of an animal, it more or less rapidly, according to the amount entering the circulation, produces first a trembling or unsteadiness of the limbs, the animal tottering, and its legs appearing stiff and partially removed from the influence of its

voluntary control. It is next unable to support itself at all on its legs; and after being affected with convulsive twitchings, is thrown into general convulsions, in which its head is forcibly drawn back, its legs outstretched, its abdomen retracted, its chest expanded and tense as a drum, and its respiration for the time suspended. Such are the visible effects of a moderately poisonous dose of strychnia, on such an animal as the dog. The paroxysms of convulsions recurring more rapidly, and being of longer duration, until finally the respiration is suspended a sufficient length of time to occasion death. Where a larger dose of strychnia is administered, the animal may have no premonitory symptoms, but may be suddenly seized with a convulsive spasm, in which it expires. Immediately after death, the muscles relax, and the extremities are flaccid; but in from half an hour to an hour's time, the *rigor mortis* presents itself to such an extent, that the extremities are rendered perfectly stiff. On the following day, I have noticed in my experiments, that a slight degree of muscular relaxation has taken place.

Many suggestions have been made as to the immediate cause of death from this energetic poison. Experiment on the lower animals, and observation on the human subject, leave no doubt that it is absorbed into the circulation, and produces its effects upon the muscular apparatus through the medium of the spinal system, leaving the functions of the brain, and probably of the sympathetic system, unimpaired. We observe the intellectual faculties remain clear in the human subject, and the effects of strychnia are manifested to an equal extent in a decapitated frog as in the unutilated animal,—showing, that the brain remains exempt from the specific effects of the poison. As long as the spinal marrow and the nerves in communication with it are intact, the tetanic condition is produced; but as soon as the former is crushed, or the latter are divided, the effects immediately subside; thus showing that it is not on the muscles or nerves, but on the spinal cord, that the spasmodic convulsions depend. The action of the heart continuing, as we shall presently see, and also the peristaltic action of the intestines, leaves it probable that the sympathetic system is unaffected.

Strychnia, then, although its visible effects are manifested



by the muscular apparatus, yet the muscles are not primarily affected, but owe their contraction to the altered functional condition of the spinal portion of the nervous system; and such muscular fibres, as those of the heart, which are not under the immediate control of the spinal system, remain uninfluenced by the poison. Whilst, therefore, the muscles of respiration, which are supplied by the spinal nerves, are firmly fixed with spasm, the muscular fibres of the heart continue alternately contracting and relaxing, provided the circulation through the lungs be maintained by artificially sustaining the respiratory process.

In reptilian animals, as the frog, where the circulation is not so dependent on the respiration as it is in the higher warm-blooded classes, the heart will continue uninterruptedly beating for many hours after life has been otherwise rendered extinct. Place, in fact, a little strychnia underneath the skin of its back, and in a few minutes the frog will become perfectly rigid with spasm; open the thorax at once, or wait some time until the animal is motionless and appears perfectly dead, and you will still find the heart beating with its ordinary regularity, as will even be the case for several hours afterwards. You may make, indeed, the heart the means of introducing the poison into the system, and it will still remain totally unaffected. I one day made an incision into the upper part of the abdomen of a frog; and after raising the sternum with a pair of forceps, snipped the pericardium, and laid a small portion of strychnine, in powder, on the surface of the heart. In four minutes the animal was rigid with spasm, but on looking at the heart it was acting as usual, as it was two hours afterwards when it was again observed, the life of the frog, in other respects, appearing extinct. I had an opportunity of showing this experiment to M. Ludovic, of Paris, and Mr. Holden, of Bartholomew's, who paid a visit to my laboratory at the time I was performing it. Where different results have been obtained (*vide* 'Lancet,' June 14th, 1856, p. 649) by applying strychnia in solution to the heart, the cessation of its action has depended on the nature of the menstruum employed. A little acetic or hydrochloric acid, with or without strychnia, will soon cause the pulsations of the heart to cease, giving it a shrunken or contracted appearance.

As strychnia produces no effect on the heart in the frog, it may be presumed that this organ has a similar immunity in the warm-blooded animal. This, however, is much less easily shown, on account of the intimate dependence of the circulation on the performance of the respiratory function. When the muscles of respiration are spasmodically fixed, a check is given to this process, which arrests the transit of the blood through the lungs, and in this way leads to a stoppage of the circulation and of the heart's action. To allow the circulation to continue, it is necessary to perform artificial respiration. But, here again we meet with a difficulty, for the muscles of the chest and belly are so fixed, that our attempts at alternately inflating and emptying the lungs, fail, unless we lay open the thoracic cavity. In the number of the 'Lancet' already referred to, Dr. Harley seeks for a satisfactory explanation of the manner in which strychnia destroys life, rejecting asphyxia from spasm of the respiratory muscles, because he did not succeed in maintaining life by means of the artificial respiration he employed. In my own experiments I have found it impossible to *succeed in performing artificial respiration*, unless the influence of the *strychnized* muscles be removed by opening the chest, and allowing the lungs to collapse to effect the expulsion of the air they contain, as in the natural expiratory action.

The two following experiments, one on a dog, the other on a rabbit, show that, if the respiration be thus sustained, the action of the heart will continue.

A middling-sized oldish mongrel bitch was placed on the table, and about a grain and a half of solid strychnia introduced under the skin of its back. In half an hour the animal trembled on its legs, and was soon unable to support itself on them. It was then seized with a convulsion, in which it was violently thrown on its side, its legs stretched out, and its head forcibly drawn back. The trachea had been previously exposed, and a large opening made into it. During the paroxysm, air seemed to issue through this opening in a series of small forcible puffs. When subsequently seized with other convulsions, its chest was noticed to be expanded, and so firmly fixed, that it appeared as tight as a drum; and its abdominal muscles were so contracted, that the parietes were

drawn in and rendered perfectly rigid. A tube connected with a pair of bellows for inflation was securely ligatured into the trachea, and artificial respiration performed. At first this succeeded in resuscitating the animal; but afterwards the paroxysm became so strong, and the chest so firmly fixed, that not the slightest impression could be made on it. Death was evidently taking place from suspended respiration. The chest was immediately opened by cutting through the cartilages of the ribs and raising the sternum, thus fully exposing the heart and lungs. The heart was beating most feebly and irregularly; but, on performing artificial respiration, which could now be easily effected, *its action became rapid and strong, and was thus kept up for twenty minutes*, when the respiration was discontinued, and the heart soon ceased. The animal, perfectly insensible when the chest was opened, afterwards appeared to regain its consciousness, and the conjunctiva retained its sensibility till the last. It was not during this time thrown into any general convulsion, but there were twitchings of the legs and of various parts of the body.

The intimate dependence of the action of the heart on the respiration was in this experiment capable of being most beautifully exemplified. Immediately the artificial respiration was for a short time suspended, the action of the heart diminished and was about to cease, when the respiration was resumed, and the heart again continued as before.

The experiment on the rabbit was similarly performed. A grain of strychnia was placed under the skin of the back, and a tube introduced and ligatured into the trachea. In five minutes the animal was under the influence of the poison, and in seven minutes was seized with a violent paroxysm, in which its chest was so firmly fixed that attempts at artificial respiration failed in producing any effect. The chest was opened, and the heart was observed beating, or rather quivering, rapidly and irregularly. The animal was still stretched out with spasm, and apparently dead to consciousness. Artificial respiration was performed, and the heart's action became normal and regular. Vitality soon seemed to return, and the animal became again sensible to external impressions which were capable of exciting a paroxysm of convulsions. The muscles of respiration, excepting during the paroxysms, acted as though they



were respiring, notwithstanding the chest was laid open, and their action was unattended with such effect. In five minutes the animal became free from spasm and insensible to external impressions, but the heart's action continued, and was maintained for ten minutes, when it ceased from the artificial respiration being stopped. On opening the abdomen the peristaltic action of the intestines was observed as usual.

It is clear, from these experiments, that strychnia has no immediate action on the heart, and that the stoppage of the circulation depends upon the arrest of the respiratory process. The spasm of the respiratory muscles that exists is all that is required to occasion death; and whether there be at the same time a spasmodic closure of the *rima glottidis*, or not, any one may easily satisfy himself that an animal will die equally as certain and as soon with a large opening in its trachea as without.

There cannot possibly be any spasm of the heart induced by the strychnia, for that would at once occasion an arrest of its action, even though the process of respiration were performed. Galvanizing the pneumogastric nerve has the effect of producing spasm of the heart; and whilst the above experiment on the dog was about, and artificial respiration was being performed, I several times transmitted an interrupted current of galvanism through one of the pneumogastric nerves, and it immediately each time occasioned, during its application, a complete arrest of the action of the heart. The heart, in fact, remained spasmodically contracted—just the same as any other muscle would do on similarly galvanizing its nerve—and could not dilate; but on discontinuing the galvanism it resumed its normal alternate contraction and relaxation.

The destruction of life by this poison, resulting from an arrest of the function of respiration, it might, *à priori*, be expected that the condition of the cavities of the heart after death would be the same as in other cases of asphyxia; namely, the right side full and the left empty, or comparatively so. This is what I have observed in my experiments; but I will give details of the three following, which were performed with especial reference to this point.

A rabbit was poisoned with a grain of strychnia dissolved in acetic acid. In two minutes, without any premonitory symptoms, it was seized with a general spasm, which jerked it off

the ground from its legs on to its side, and from which it did not recover. It was placed aside till the following day, when the chest was opened and the heart examined. The right cavities were rather distended with coagulated blood. The left auricle was full but not distended, and the left ventricle contained a very small quantity of blood.

One-twentieth of a grain of solid strychnia was administered to a half-grown rabbit. In eighteen minutes it manifested symptoms, and in twenty it died. The heart on the following day presented precisely the same appearance as the one just alluded to.

One-fiftieth of a grain of solid strychnia was given to a rabbit, likewise half-grown. After sixteen minutes it manifested symptoms of poisoning, but subsequently completely recovered. On the following day one-thirtieth of a grain was given to it. Symptoms appeared in fourteen minutes, and in thirty-five minutes it died. On examining the heart, the day after, the cavities on the right side were full; the left auricle contained a little blood, and the left ventricle none.

In the recent notorious case of Cook, great stress was laid on the empty condition of the heart. As, however, death of the heart is only a secondary result, its condition will depend upon the precise mode of termination of the respiration. In exceptional cases I can conceive it possible that after the muscles have relaxed from the death-spasm, a few automatic efforts at respiration may be performed, which are sufficient to urge onwards the circulation through the lungs, and empty the right cavities of the heart, although insufficient to stimulate the oppressed brain to the resuscitation of life.

### WOORALI POISON.

For some of this scarce, poisonous material, I am indebted to Mr. Waterton, who many years ago undertook an excursion a considerable distance into the interior of South America, to obtain possession of it from the natives, and to ascertain precise facts with regard to its nature. It is in the form of a dry extract, of a brownish colour, and easily soluble in water.

If an incision be made through the skin of the back of a frog, and a small portion of the poison be placed underneath; in

eight or nine minutes it will appear weak on its legs, and in a minute or two more its extremities will be flaccid. All its voluntary muscles are now paralysed, and it is therefore totally incapable of moving or exhibiting any signs of consciousness. It appears, indeed, like a frog that has been dead for some time; but if its thorax be opened, its heart will be found beating as regularly as under ordinary circumstances, and will continue so for many hours. Like strychnia, the Woorali poison has no effect on the heart; but it is diametrically opposed in its action on the voluntary muscles; producing paralysis instead of spasm.

It is a most interesting fact, that a single experiment is sufficient to show that the paralyzing effects of the Woorali depend upon a destruction of nervous energy, and not upon a loss of muscular power. We are told by some physiologists that a muscular power *sui generis* does not exist, but that the force of muscular contraction is derived from the nervous system. Now, an experiment with this poison at once contradicts this doctrine, and establishes irrefutable evidence of a force inherent in the muscles themselves. If a frog be killed by decapitation or any other ordinary method, and the crural nerves exposed, an interrupted current of galvanism immediately excites a powerful movement of the extremities. If, however, the frog be poisoned with the Woorali, and the crural nerves be similarly exposed, no amount of galvanism or of mechanical irritation is capable to exciting the slightest muscular movement; but if the galvanism be applied to the muscles themselves, an immediate and strong contraction takes place.

The following are the details of an experiment on a dog, with the Woorali poison, in which the heart's action was maintained by artificial respiration, and which the students of Guy's will remember my performing before them on the theatre table, after one of my lectures during the summer session.

A four or five months' pup was inoculated with the poison, by introducing a small quantity in solution under the skin of the back. In about twenty minutes' time it began to appear weak on its legs, which soon gave way under it, when it attempted to walk or stand. It now lay unable to get up, and,



as was to be expected, never manifested the slightest sign of spasm or convulsion; indeed, its legs were perfectly flaccid. Its respiration becoming difficult and slowly performed, a tube was ligatured into the trachea, and artificial respiration resorted to, which was easily accomplished, on account of the free mobility of the chest. When all power of natural breathing had ceased, and the animal appeared dead, the chest was laid open and the heart exposed. Artificial respiration being maintained, the action of the heart was kept up, as in the case of the experiment with strychnia, for full twenty minutes, when it was allowed to cease. An interrupted current of galvanism applied to the pneumogastric nerve had no power, like in the dog under the influence of strychnia, of checking the action of the heart by inducing spasm; because, as we have seen, the capacity of the nervous system was entirely destroyed by the influence of the poison.

LIST  
OF  
GENTLEMEN EDUCATED AT GUY'S HOSPITAL,  
WHO HAVE PASSED THE  
EXAMINATIONS OF THE SEVERAL COLLEGES, UNIVERSITIES,  
&c. &c.  
*Since September, 1855.*

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*Members of the Royal College of Surgeons of England.*

OCTOBER, 1855.  
John Winter Dryland.

NOVEMBER.

Thomas Blasson.		John Mustard.
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DECEMBER.

William Elphick.		Charles Lloyd Morrice.
George Browning.		Silvanus Jones Morris.
Thomas Carter.		William James Harris.

JANUARY, 1856.

William Jay.		Henry Gibbons.
Richard Coles.		Henry Penfold.
Philip William Freshfield.		Daniel Shelswell.
Edmund Lloyd.		

MARCH.

William Unwin.		Thomas Roberts.
Samuel Giles.		Francis Scott.
William Venour.		Edmund Wm. Valentine.
William Renne Bennett.		William Frederick Brook.
Francis Pool Lansdown.		John James Nason.
Edward Henry May.		

APRIL.

Samuel Best A. Denton.  
Charles Sprigg.  
John Henry Tuke.  
James Foster Gray.  
George Dawson Hunt.  
William Shillinglow Hewat.

Peter Herbert Desvignes.  
John Irwin.  
William Alfred Skinner.  
William Brown Arminson.  
Frederick Piercey.

MAY.

John Edward Barnes.  
John Caparn.  
Charles Pigg.  
George William Daniell.  
Sylvester R. Skinner.

Edward Bachelor Terry.  
Edward Francis Weston.  
William Sowerby Wallen.  
George Theophilus Jepson.

JUNE.

Francis Clowes.

Charles Evans Muriel.

JULY.

Robert Arthur Elliott.  
Robert Ratheron Stilwell.

Charles George Leonard.  
George Robert Adcock.

AUGUST.

Mr. Frederick Cope Bartlett.

**Licentiates in Midwifery.**

John Winter Dryland.  
Henry Gibbons.  
Richard Coles.  
Edmund Lloyd.  
Charles Lloyd Morrice.  
Edmund W. Valentine.  
Thomas Asslin.  
Henry Penfold.  
Daniel Shelswell.  
Samuel Giles.

Owen Davies.  
Edward Henry May.  
Charles Pigg.  
S. R. Skinner.  
E. B. Terry.  
W. S. Wallen.  
Henry Watson.  
Charles E. Muriel.  
Richard U. Wallace.

**Licentiates of the Apothecaries' Society.**

AUGUST, 1855.

Thomas G. Wales.



OCTOBER.

Thomas Carter.

NOVEMBER.

Edwin Jones.

Charles Nottidge.

Henry Jones.

DECEMBER.

William Frederick Brook.

JANUARY, 1856.

Edward Henry May.

Charles George Leonard.

Edward B. Terry.

Edward F. Weston.

FEBRUARY.

William S. Wallen.

George W. Daniel.

Robert R. Stilwell.

James B. Baker.

Charles Sprigg.

Eugene A. Kingsley.

Allen Chattaway.

William Elphick.

John James Nason.

MARCH.

Henry Hayes Crawford.

Owen Davies.

John Mustard.

Charles E. Muriel.

William H. Aldersey.

Uriah P. Brodribb.

Sylvester R. Skinner.

Francis P. Lansdown.

APRIL.

William F. Dix.

Charles John Fluder.

David W. Trimmell.

Charles J. Watson.

George D. Hunt.

William B. Arminson.

Alfred B. Duffin.

James F. Gray.

Henry Kelsall.

William Powell.

William L. Le Sage.

MAY.

Owen Tucker.

John Henry Tuke.

JUNE.

William Alfred Skinner.

William Blason.

William Freeland Johnson.

Francis Scott.

JULY.

Francis Clowes.

Frederick Duffy.

William R. Bennett.

AUGUST.

Thomas Blason.

**University of London.**

**SECOND EXAMINATION FOR BACHELOR OF MEDICINE, 1856.**

- \* William Tiffin Iliff . . . . First Division.  
George Mayris Pittock . . . Second Division.

- \* Obtained the Gold Medal in Surgery, and placed third in Medicine.

**FIRST EXAMINATION FOR BACHELOR OF MEDICINE, 1856.**

- \* William Hodges . . . First Division.  
William H. Aldersey } Second Division.  
James Proctor }

- \* Obtained the Gold Medal for Materia Medica.

**University of St. Andrew's.**

OCTOBER, 1855.

John Ince.  
Edwin Jones.  
Frederick Martin.  
Richard H. Wilbe.

MAY, 1856.

William B. Arminson.  
Charles Sprigg.

**Royal College of Physicians, London.**

Samuel O. Habershon . . . .	}	Fellows of the Royal College.
Samuel Wilks . . . .		
William Odling . . . .	}	Licentiates.
Frederick W. Pavy. . . .		

**East India Company's Service.**

BY EXAMINATION.

William D. Trimnell.  
Newton Thomas Brigstocke.

Alexander M. Dallas.  
Arthur Kelsey.

**GENTLEMEN WHO HAVE HELD THE APPOINTMENT OF  
HOUSE SURGEONS.**

John Caparn.  
Richard U. Wallace.  
Edmund W. Valentine.

GENTLEMEN TO WHOM DRESSERSHIPS HAVE BEEN AWARDED.

Edward Hy. May.	Arthur D. Brooks.
William Venour.	Robert R. Stilwell.
G. H. Dyer.	John J. Nason.
W. F. Johnson.	Chas. E. Muriel.
R. U. Wallace.	E. B. Terry.
J. F. Gray.	Robert Hicks.
E. F. Weston.	G. T. Jepson.
E. W. Valentine.	W. F. Bennet.
William Holmes.	Fred. Hy. Smith.
Geo. W. Daniell.	Charles Pigg.
S. R. Skinner.	Francis Clowes.
S. Giles.	W. F. Dix.
U. P. Brodribb.	W. B. Arminson.
Thos. Blasson.	C. J. Watson.
John Hy. Tuke.	

PUPIL'S PHYSICAL SOCIETY'S PRIZE.

Sylvester R. Skinner.

GENTLEMEN WHO FILLED THE POST OF CLINICAL CLERK DURING  
THE WINTER SESSION OF 1855-6.

Henry Gibbons.	Charles Evans Muriel.
Samuel Giles.	John Henry Tuke.
Sylvester R. Skinner.	Edward B. Terry.
Robert R. Stilwell.	Arthur D'Oyly Brooks.

*Summer Session, 1856.*

G. H. Dyer.	W. L. Baker.
Geo. Jepson.	E. W. Valentine.
William Venour.	W. B. Arminson.

RESIDENT OBSTETRIC CLERKS SINCE SEPTEMBER 1, 1855.

SEPTEMBER, 1855.

William Hall . . . .	Senior Clerk.
Thomas Asslin . . . .	Second Clerk.
John Henry Tuke . . . .	Junior, 1st Fortnight.
Charles Evans Muriel . . . .	Junior, 2d ditto.



## OCTOBER.

Thomas Asslin	.	.	Senior Clerk.
Thomas T. Gardner	.	.	Second Clerk.
William Holmes	.	.	Junior, 1st Fortnight.
William A. Skinner	.	.	Junior, 2d ditto.

## NOVEMBER.

Henry Gibbons	.	.	Senior Clerk.
Arthur Kelsey	.	.	Second Clerk.
W. F. Johnson	.	.	Junior, 1st Fortnight.
W. F. Dix	.	.	Junior, 2d ditto.

## DECEMBER.

John James Nason	.	.	Senior Clerk.
Charles Evans Muriel	.	.	Second Clerk.
Frank Clowes	.	.	Junior, 1st Fortnight.
George Jepson	.	.	Junior, 2d ditto.

## JANUARY, 1856.

Serjeant J. C. Norman	.	.	Senior Clerk.
Charles Lloyd Morrice	.	.	Second Clerk.
Sylvester Rutherford Skinner	.	.	Junior, 1st Fortnight.
Arthur D'Oyley Brooks	.	.	Junior, 2d ditto.

## FEBRUARY.

Charles Lloyd Morrice	.	.	Senior Clerk.
Newton T. Brigstocke	.	.	Second Clerk.
G. H. Galton	.	.	Junior, 1st Fortnight.
Richard Coles	.	.	Junior, 2d ditto.

## MARCH, 1856.

Newton T. Brigstocke	.	.	Senior Clerk.
Edward B. Terry	.	.	Second Clerk.
David S. Skinner	.	.	Junior, 1st Fortnight.
W. C. Nicholas	.	.	Junior, 2d ditto.

## APRIL.

Edward B. Terry	.	.	Senior Clerk.
Edward Francis Weston	.	.	Second Clerk.
Joseph Littlewood	.	.	Junior, 1st Fortnight.
W. L. Baker	.	.	Junior, 2d ditto.

## MAY.

Edward Francis Weston	.	.	Senior Clerk.
William Frederick Dix	.	.	Second Clerk.
C. H. Carver	.	.	Junior, 1st Fortnight.
Pio Rengifo	.	.	Junior, 2d ditto.

JUNE.

William Frederick Dix	.	.	Senior Clerk.
William Venour	.	.	Second Clerk.
James Palfrey	.	.	Junior, 1st Fortnight.
Frederick Henry Smith	.	.	Junior, 2d ditto.

JULY.

John Henry Tuke	.	.	Senior Clerk.
George Williamson Daniell	.	.	Second Clerk.
Henry C. Biddle	.	.	Junior, 1st Fortnight.
Alexander M. MacDougal	.	.	Junior, 2d ditto.

AUGUST.

George Williamson Daniell	.	.	Senior Clerk.
Sylvester Rutherford Skinner	.	.	Second Clerk.
James Broad	.	.	Junior, 1st Fortnight.
James Emptage Moore	.	.	Junior, 2d ditto.

---

NUMBER OF CASES ATTENDED DURING THE YEAR ENDING  
AUGUST, 1856.

September, 1855	.	.	.	.	150
October	.	.	.	.	118
November	.	.	.	.	145
December	.	.	.	.	186
January, 1856	.	.	.	.	156
February	.	.	.	.	172
March	.	.	.	.	198
April	.	.	.	.	168
May	.	.	.	.	160
June	.	.	.	.	159
July	.	.	.	.	107
August	.	.	.	.	166
Total	.	.	.	.	1885

---

HONORARY OBSTETRIC CERTIFICATES

Awarded since October, 1855, for attending above 100 cases during Twelve Months.

W. L. Baker.  
E. F. Weston.  
C. H. Carver.

James Palfrey.  
James Emptage Moore.

## ADJUDICATION OF THE ASTLEY COOPER TRIENNIAL PRIZE.

YEAR.	SUBJECT.	SUCCESSFUL CANDIDATE.	WHERE PUBLISHED.
1844.	On the Structure and Use of the Thymus Gland.	John Simon, Esq. F.R.S.	London, Henry Renshaw.
1847.	On the Structure and Use of the Renal Capsules.	Richard Halahan, Esq.	Unpublished.
1850.	On the State of the Blood and Blood-vessels in Inflammation.	T. Wharton Jones, Esq., F.R.S.	Guy's Hospital Reports, series ii, vol. 7, p. 1.
1853.	On the Structure and Use of the Spleen.	Henry Gray, Esq., F.R.S.	London, John W. Parker and Son.
1856.	On the Cause of the Coagulation of the Blood.	B. W. Richardson, M.D.	



## ASTLEY COOPER PRIZE.

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THE Condition annexed by the Testator is, “That the Essays or “Treatises written for such Prize shall contain original experiments “and observations, which shall not have been previously published; “and that such Essays or Treatises shall (as far as the subject shall “admit of) be illustrated by preparations and drawings, which preparations and drawings shall be added to the Museum of Guy’s “Hospital, and shall, together with the Work itself, and the sole and “exclusive interest therein and the copyright thereof, become thenceforth the property of the Hospital, and be transferred as such by “the successful candidate.”

It is the will of the Founder that no Physician, or Surgeon, or other officer for the time being, of Guy’s Hospital or of St. Thomas’s Hospital, nor any person related by blood or affinity to any such Physician, or Surgeon, or other officer for the time being, shall at any time be entitled to claim the Prize; but, with the exception here referred to, this (the Astley Cooper) Prize is open for competition to the whole world.

Candidates are informed that their Essays, either written in the English Language, or, if in a Foreign Language, accompanied by an English translation, must be sent to Guy’s Hospital on or before January 1st, 1859, addressed to the Physicians and Surgeons of Guy’s Hospital.

Each Essay or Treatise must be distinguished by a Motto, and be accompanied by a sealed envelope containing the Name and Address of the writer. None of the envelopes will be opened, except that which accompanies the successful Treatise. The unsuccessful Essays or Treatises, with the illustrative preparations and drawings, will remain at the Museum of Guy’s Hospital until claimed by the respective writers or their agents.

# G U Y ' S.

1856-7.

## THE MEDICAL SESSION

COMMENCES ON THE FIRST OF OCTOBER.

THE INTRODUCTORY ADDRESS will be given by Thomas Bell, F.R.S., and Pres. L.S., on Wednesday, the First of October, at Two o'clock.

Gentlemen desirous of becoming Students must produce satisfactory testimony as to their Education and Conduct; they are required to pay £40 for the first year, £40 for the second year, and £10 for every succeeding year of attendance. One payment of £100 entitles a Student to a perpetual Ticket.

The Payment for the year admits to the Lectures, Practice, and all the privileges of a Student for that year only.

Clinical Clerks, Dressers, Ward Clerks, Dressers' Reporters, Obstetric Residents, and Dressers in the Eye Ward, are selected according to merit from those students who have attended a second year.

Every Student is required to conform to the Rules and Regulations for the internal management of the Hospital.

The privileges of a Student will be withdrawn in the event of neglect or misconduct.

Certificates will not be given for Lectures, and Practice, unless duly attended.

The Christmas Recess commences December 24th. The Lectures are resumed January 6th. The Winter Session terminates March 31st. The Summer Course commences May 1st, and concludes July 31st.

## MEDICAL OFFICERS.

*Consulting Physician.*—RICHARD BRIGHT, M.D., F.R.S.

*Physicians.*—THOMAS ADDISON, M.D.; G. H. BARLOW, M.D.; H. M. HUGHES, M.D.; OWEN REES, M.D., F.R.S.

*Assistant Physicians.*—W. W. GULL, M.D.; S. O. HABERSHON, M.D.; S. WILKS, M.D.

*Surgeons.*—EDWARD COCK, ESQ.; J. HILTON, ESQ., F.R.S.; J. BIRKETT, ESQ.

*Assistant Surgeons.*—ALFRED POLAND, ESQ.; THOMAS CALLAWAY, ESQ.; J. COOPER FORSTER, ESQ.

*Obstetric Physicians.*—J. C. W. LEVER, M.D.; HENRY OLDHAM, M.D.

*Surgeon Dentist.*—T. BELL, ESQ., F.R.S. and PRES. L.S.

*Surgeon of the Eye Infirmary.*—JOHN F. FRANCE, ESQ.

*Apothecary.*—JAMES STOCKER, ESQ.

## LECTURES, &c.

### WINTER COURSES.

*Medicine.*—DR. OWEN REES and DR. GULL, Mondays, Wednesdays, and Fridays, at half-past three.

*Clinical Medicine.*—DR. ADDISON, DR. BARLOW, DR. HUGHES, and DR. OWEN REES.

*Surgery.*—MR. HILTON and MR. BIRKETT, Tuesdays, Thursdays, and Saturdays, at half-past three.

*Clinical Surgery.*—MR. COCK, MR. HILTON, and MR. BIRKETT.

*Anatomy.*—MR. POLAND and DR. PAVY, daily at Two, (except Tuesdays).

*Physiology.*—DR. PAVY, Tuesdays, Thursdays, and Saturdays, at nine.

*Demonstrations on Anatomy.*—MR. J. COOPER FORSTER, and MR. BURTON BROWN, daily.

*Demonstrations on Morbid Anatomy.*—DR. WILKS, daily, at half-past two.

*Clinical Lectures on Midwifery and Diseases of Women.*—DR. LEVER and DR. OLDHAM.

*Chemistry.*—DR. ALFRED S. TAYLOR, Tuesdays, Thursdays, and Saturdays, at eleven.

*Moral Philosophy.*—THE REV. T. H. BULLOCK, M.A., Chaplain to the Hospital.

*Experimental Philosophy.*—DR. ODLING and MR. BURTON BROWN, Wednesdays, at eleven.

### SUMMER COURSES.

*Demonstrations on Cutaneous Diseases.*—DR. ADDISON and DR. GULL, Mondays, at one.

*Materia Medica.*—DR. HABERSHON, Tuesdays, Thursdays, and Saturdays, at two.

*Clinical Medicine.*—DR. GULL, DR. HABERSHON, and DR. WILKS.

*Clinical Surgery.*—MR. POLAND, MR. CALLAWAY, and MR. J. COOPER FORSTER.



*Midwifery*.—DR. LEVER and DR. OLDHAM, daily, at a quarter to nine.

*Dental Surgery*.—MR. SALTER.

*Medical Jurisprudence*.—DR. ALFRED S. TAYLOR, Tuesdays, Thursdays, and Saturdays, at ten.

*Ophthalmic Surgery*.—MR. FRANCE, Wednesdays and Fridays, at three.

*Pathology*.—DR. WILKS.

*Comparative Anatomy*.—DR. PAVY.

*Botany*.—MR. JOHNSON, Tuesdays, Thursdays, and Saturday, at half-past eleven.

*Practical Chemistry*.—DR. ODLING, Mondays, Wednesdays, and Fridays, ten to two.

*Pupils' Physical Society*.—Saturdays, at seven in the evening.

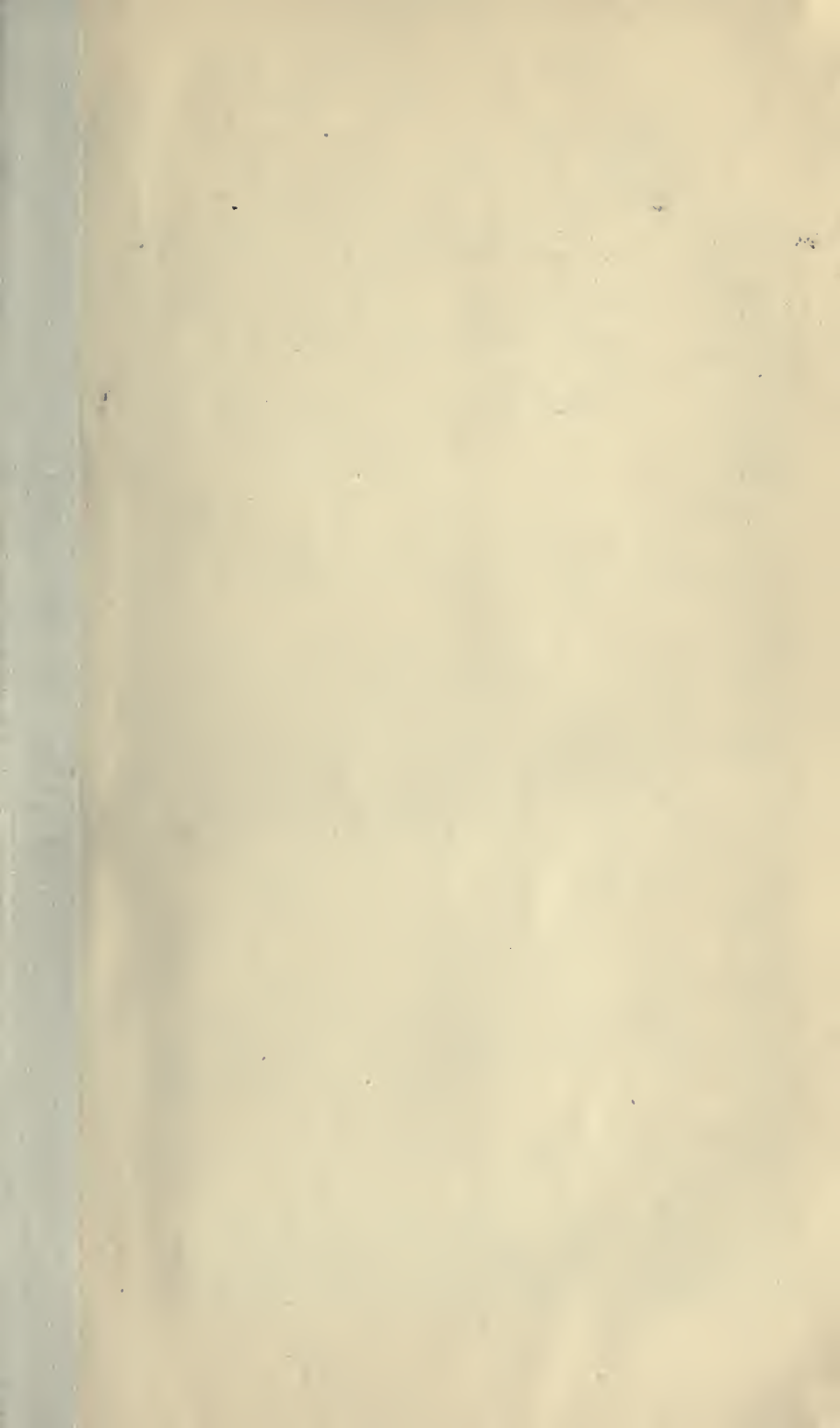
*The Clinical Wards* will open the first week in October and first week in May.

*Lying-in Charity*.—DR. LEVER and DR. OLDHAM.

*Curator of the Museum*.—DR. WILKS.

THE LIBRARY, MUSEUMS, AND MODEL ROOMS, ARE OPEN DAILY TO THE STUDENTS, FROM NINE O'CLOCK A.M., TILL FIVE O'CLOCK P.M.

MR. STOCKER, *Apothecary to Guy's Hospital*, is authorized to enter the Names of Students.



1897. 43  
1898. 44

1899. 45



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By ALFRED PAIRPOINT.

DEDICATED BY PERMISSION TO HIS EXCELLENCY, THE HONOURABLE  
G. M. DALLAS, Esq., AMERICAN AMBASSADOR TO ENGLAND.

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